POST-GRADUATE LECTURES
ON
ORTHOPEDIC DIAGNOSIS
AND
INDICATIONS

By

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VOLUME II

SECTION A Paralytic Disabilities
SECTION B Static Disabilities



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INTRODUCTION

PARALYTIC diseases are of interest to the orthopedic surgeon in all their phases, and this applies foremost to the paralytic disabilities due to poliomyelitis It is proper that the internist or pediatrician take charge in acute stages when the disease is still in its systemic manifestations This should not mean a sharp division of responsibilities It is not a matter of "taking over' at a certain point when residual deformities engage the attention of the orthopedist exclusively It is much more a case of sharing responsibilities with the internist from the start Diagnosis and treatment of residual deformities requires a sound knowledge of etiological and pathological facts which precede the development of these deformities We feel strongly that the bounds of these orthopedic lectures are not being overstepped by according a due share to the presentation of etiological and pathological events which concern the earlier and more acute stages of the disease

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POST-GRADUATE LECTURES
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SECTION A

THE PARALYTIC DISABILITIES

Lecture I

ON POLIOMYELITIS (PATHOGENESIS AND PATHOLOGY)

I PATHOGENESIS

A THE VIRUS, ITS PATHWAY AND LOCALIZATION

THE virus of poliomyelitis is a globulin body whose host range is limited to the monkey and the cotton rat That it is the cause of poliomyelitis has been shown by Landsteiner and Popper,[11] it was isolated in brain and cord by Flexner and Noguchi, it was successfully transmitted in monkeys via the intracerebral route by Leiner and Wiesner [12]

That a real systemic infection precedes its localization in the spinal cord is shown by the clinical picture In many cases there is an initial rise of temperature, followed by an intermission and then by a second rise which ushers in symptoms of the central nervous system Yet, experimental transmission of the virus through the blood stream fails in monkeys because the chorio-endothelial system is a barrier which separates the central nervous system from the general circulation.

How then, does the virus reach the cord and brain? Our present concept is that it does so by way of the axis cylinders (Fairbrother and Hurst[7]), wandering along the peripheral nerve through the axioplasm of the axons The virus is, in other words, not only neurotropic, i e , bound to the central nervous system, but also neuronotropic, i e., bound to the pathway of axis cylinders In fact, interruption of the nerve fibers by cutting or freezing can prevent inoculation (Bodian and Howe, H A [2])

B THE MECHANISM OF INFECTION

The transmission of the virus from patient to patient occurs by extraneous route Uterine transmission is unknown (H M Weever, G Steiner, H Ammon and M Hastings[20]) Congenital poliomyelitis therefore does not exist Consequently, the child can receive no immunity even if born of a poliomyelitic mother

C THE PORT OF ENTRY

In experiments on the monkey the most convenient way of inoculation is by the intranasal route This runs from the olfactory bulbs through the preoptic area to the hypothalamus and then descends downward to the medulla and the cord In man this is not the usual route of infection It is now believed that the most important port of entry is the gastrointestinal tract, especially its upper portion, the mouth, pharynx and esophagus It has been found that the

incidence of bulbar and bulbospinal poliomyelitis is higher in patients who have their tonsils and adenoids removed, and such removal should be avoided during the poliomyelitis season Toomey and Krill[18] cite a case of tonsillectomy performed on the same day on five apparently healthy children in one family All five developed bulbar poliomyelitis and three of them died In the alimentary tract the virus becomes fixed in the unmedullated post ganglionic fibers of the thoracolumbar outflow and spreads centripetally from the sympathetic system to the somatic nerves It reaches the cord through the anterior roots by way of the preganglionic fibers and invades the anterior horn cells It may travel along the sympathetic chain to reach the cervical region While it is true that the upper portion of the alimentary tract, pharynx, mouth and esophagus is most often the port of entry, there is also evidence that all of the gastrointestinal tract may be the primary source since the virus has been recovered from the stools of monkeys as well as from the stools of patients (Trask, Paul and Melnik[19])

It is also not unlikely that the skin is a portal of entry, which raises the point that the infection may be transmitted by insects Filtrates of bugs fed upon infected monkeys were able to produce poliomyelitis when injected intra cerebrally and flies collected within an epidemic area were found positive for the virus (Trask Paul and Melnik[19]) Of further interest is the fact that poliomyelitis is not only transmitted from patient to patient but also by healthy carriers (H. A Wenner and A E Casey,[21] McClure and A. D Langmuir[13]) In some cases of poliomyelitis the infection is so mild that it escapes attention and it is not diagnosed. It is therefore difficult to distinguish between healthy carriers and convalescent carriers

D THE PORT OF EXIT

As the gut serves as a reservoir for the secretions of the upper respiratory and alimentary tract, the virus is excreted in the human feces and has been found repeatedly in sewage There seems to be no evidence that the virus is excreted in the urine or through the skin

E IMMUNITY

We must assume that man possesses a high natural immunity against the disease because only 6 per cent of all individuals exposed to poliomyelitis will acquire it even of this number only a comparatively small percentage becomes actually paralyzed It is believed that one attack provides sufficient and lasting immunity even if no paralysis develops Up to date only 13 authenticated cases of a second attack are reported in the literature we observed two in our own clinic (L Cohen[6]) The susceptibility to the disease is greatly increased in childhood and in young adults It decreases after middle age.

II PATHOLOGY

A CHANGES OCCURRING IN THE CENTRAL NERVOUS SYSTEM

1 The changes within the cord

It was Charcot who identified the disease as an involvement of the motor ganglion cell of the spinal cord The affected anterior horn cell shows first a central chromatolysis (Fig 1) Then follows a process of fragmentation and finally a breaking up of the cell, the so-called satellitosis (Fig 2) It ends with complete destruction and absorption of the cell by phagocytosis Secondary to these cell changes one observes an interstitial inflammation manifested by peri

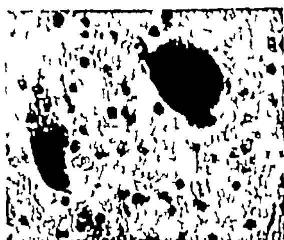


Fig. 1 Chromatolysis of anterior horn cell.

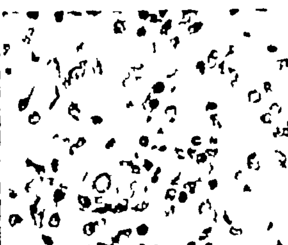


Fig. 2 Fragmentation and satellitosis of anterior horn cell.

vascular infiltration of leukocytes, the so-called perivascular cuffing (Fig 3) While the destruction of the ganglion cell represents a permanent loss of motor function, the interstitial inflammation is transitory This explains the recession of symptoms and particularly the decrease of paralysis following the acute stage

Still up to a certain point, changes in the ganglion cells must be considered reversible (Cadwalader[4] and Wickman[22]) Early swelling and chromatolysis may be so considered while the later changes of fragmentation and satellitosis are obviously irreversible Reversible changes in form of chromatolysis and peripheral arrangement of the Nissl bodies can still be found within one week following the onset after one month however, such reversible changes in the anterior horn have almost entirely disappeared (Bodian[1, 2]) The lateral horn cells usually escape destruction and there are few changes seen in the posterior horn except typical inflammatory cells (Fig 4), although some lesions are found in Clarke s column Regarding the white matter of the cord, mild and diffuse demyelinization is observed in the ventral and lateral columns but no changes in the pyramidal tract.



FIG. 3 Perivascular "cuffing.

FIG. 4 Demyelinization and inflammatory perivascular infiltration in posterior column.

QUADRICEPS
TIBIALIS ANTICUS
BICEPS (THIGH)
TIBIALIS POSTICUS
PERONEAL
GASTRO SOLEUS
after A. SANO

FIG. 5 Sano's scheme of distribution in the anterior horn of motor nuclei for the individual extremity muscles.

The striking feature in the pathology of poliomyelitis is the selectiveness of the paralysis and the prevalence of the partial over the total paralysis of the muscles, a point to which Lovett had already called attention There are discrete regional destructions in spots so that, as a rule, not all or not even a major part of the cells of the column are involved, except in severe cases of total paralysis

The adventitious motor clusters which normally appear in the lateral portion of the anterior horn at the cervical or lumbar intumescence are due to the



Fig. 6 Chromatolysis and satellitosis of internuncial cells.



Fig. 7 Inflammatory infiltration in cerebral cortex.

addition of new muscle units in the extremities, the clusters representing the motor nuclei of the nerves supplying these muscles They are arranged in such a fashion that the more distal muscle groups have their nuclear centers located more and more along the lateral contour of the anterior horn (Fig 5)

The changes in the anterior horn cells are not the only ones in the gray matter Similar changes are seen in the so-called internuncial cells, whose function is to act as a synapsis between the fibers of the cord and the motor horn cells These cells are arranged in clusters which lie behind and medially to the anterior horn cells They are similar to the latter in appearance except that their Nissl bodies are smaller (Fig 6) The internuncial cells are often found destroyed together with the anterior horn cells It is the accepted opinion that the internuncial cells exercise an inhibitory action on the muscle tone, and that the lesion of this cell group produces a hypertonus of the skeletal muscles All impulses going through the spinal reflex arc as well as those coming from the higher centers must be relayed through the internuncial cells to the motor neurons of the anterior horn Much speculation has arisen regarding the

pathological equivalent of pain and skin irritability The spinal ganglia, for instance, have been found at autopsy to be involved (Jonnesco[10]) in the territory which corresponded to the paralyzed region, and foci of infiltration of the dorsal root have been recognized for decades (B Sachs and Strauss[11])

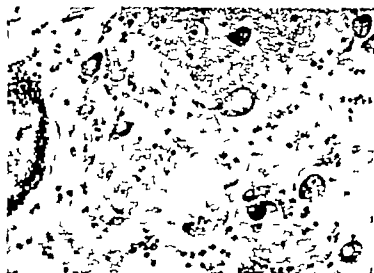


Fig. 8. Inflammatory changes in floor of fourth ventricle.



Fig. 9 Inflammatory infiltration in meninges.

2. Changes in the central nervous system outside the cord

Of special interest are the changes in the cortex located primarily in the paracentral gyrus and consisting of the perivascular collars of lymphoid cells (Fig 7) Similar inflammatory infiltrations are noted in the fourth ventricle (Fig 8) in the midbrain and even in the meninges (Fig 9) On the whole, one finds that in cranial direction from the medulla oblongata upward the changes are mainly interstitial while the main seat of the parenchymatous lesion still remains the anterior horn cell group

B CHANGES OUTSIDE THE CENTRAL NERVOUS SYSTEM

1 Outside the locomotor system

Visceral changes have been variously reported in the myocardium, in the gastro intestinal tract mucosa and in the mesenteric lymph glands (Saphir[14])

2 In the locomotor system

The muscle changes are secondary to the involvement of the central nervous system and consist in atrophy, degeneration and disintegration and, finally, replacement (Hipps) Some muscles show more homogenous atrophy and degeneration, others, more fatty infiltration or fibrous replacement Homogenous atrophy occurs, especially in the small intrinsic muscles (Hipps[9])



Fig. 10 Paralytic muscle spotty atrophy

The irregular and spotty character of muscle atrophy and degeneration is of interest We find complete degeneration with fibrous substitution, together with intact muscle bundles in the same microscopic field (Fig 10)

In the neuromuscular end plate inclusion bodies appear after denervation, and the end plate becomes pyknotic (Cary[3]) The disintegration and disconnection of the myoneural junction of the muscle fibers is complete within 36 hours On the other hand, the sensory muscle spindle acts differently It is more resistant during the early stage of the disease, but some changes are noticeable They might be the anatomic corollary of pain and tenderness, as well as of myotatic reflex rigidity

III EXPERIMENTAL PATHOLOGY AND PATHOPHYSIOLOGY OF POLIOMYELITIS

Experimental studies on denervated muscles have revealed some valuable facts relative to the nature of muscle regeneration

A THE EFFECT OF VITAMINS ON REGENERATION

No effect of vitamins on regeneration has been definitely established (H M Hines[8]), although it was found that the regeneration of muscles in animals subsisting on a low vitamin C diet was relatively slower than in others with adequate diets Vitamin B_2 or riboflavin as well as vitamin B_1 or thiamin deficiency, while producing a general drop in weight, muscle weakness and loss of reflexes, has no specific effect upon a denervated muscle Neither does vitamin E deficiency affect neuromuscular regeneration

B THE EFFECT OF IMMOBILIZATION ON REGENERATION

It seems that the *lack of immobilization* of a partially denervated muscle does not interfere with regeneration of the motor nerve supply In fact, regeneration was found to be more complete in animals which had no immobilization at all, and it would seem therefore that immobilization retards rather than enhances regeneration of the partially denervated muscle.

It is well to draw a distinction between simple atrophy and degeneration. The latter is the more irreversible pathological change The atrophied muscle does not exhibit the characteristic degeneration, a fact to which Ridlon called attention many years ago The atrophic muscle can recover even in advanced stages, a muscle in advanced stages of degeneration does not recover Simple atrophy shows shrinkage of the fibers without a real increase in sarcolemma nuclei, there is disappearance of longitudinal and cross striation, these changes are still reversible In degeneration, however, there is vacuolization, necrosis, crumbling of the sarcoplasm proliferation of interstitial tissue and finally fibrosis or fatty substitution These changes are irreversible.

C THE ELECTROPATHOLOGY OF POLIOMYELITIS

In the interpretation of some of the clinical phenomena, electrodiagnosis has been of greatest value in two investigative lines The chronaxia and the action current.

1 The chronaxia

It has been known for a long time that neuromuscular degeneration can be determined by measuring the chronaxia that is, the time necessary for an electrical stimulus of known and standardized intensity to produce a muscular response The chronaxia values for the paralyzed muscle have been studied particularly by Moldaver,[14] who has found that while the normal chronaxia time is from 0 6 to 1 5 sigma in partial paralysis after injury to the motor nerve the chronaxia time may be 120 times the normal and more, this is in contrast to the spastic muscle where the lower motor neuron is intact and where there is no such increase in chronaxia

It will be recalled that under the Kenny concept the paralyzed muscle is the so-called alienated muscle and is supposedly not affected, while the spastic muscle is believed to be the affected one. Now it has been found that this *so-called alienated muscle has at times a chronaxia 100 times greater than that* of the normal muscle, whereas the chronaxia in the so-called affected spastic muscle is normal The spasm which is one of the foremost clinical symptoms is believed to be at least in part, the clinical corollary of lesions of the internuncial cell group This group is a synaptic link connected with the inhibiting motor impulses from higher centers However spasm has other sources It may be due to meningeal irritation or irritation of posterior root ganglia, or be a reflex from sensory or proprioceptive impulses The principal point is that it does not lead to neuromuscular degeneration (Moldaver[14])

2 The action current

Trying to find an explanation for the spasm, R P Schwartz and Bouman[17] were able to show that muscle spasm exists not only in the antagonist of the paralyzed muscle but also in the weakened muscle itself and even in muscles which otherwise show no clinical signs at all and which are situated in more remote parts of the body Spasm is, therefore, not a lower motor neuron sign at all, but it is of reflex nature, it may be superimposed upon a normal muscle so strongly that the contractility may become entirely submerged for the time being However, as voluntary contraction increases, spasm decreases Furthermore, it is entirely a transitory symptom though for the time being it may be the dominant sign

Schwartz and Bouman[17] also observed action currents in the affected muscle corresponding to the degree of spasm This means a hypersensitivity to stretching an increased stretch reflex It is significant that in contractures which are due to intrinsic muscle shortening there is no action current and the muscle is entirely inert, as is also the completely paralyzed muscle There is additional experimental evidence from the study of action currents (M A Brazier, A L Watkins and R S Schwab) that a partially paralyzed muscle on stretching may become hyperirritable or show increased stretch reflex and that such a paretic, hyperirritable muscle will discharge electric potential even at rest. Furthermore there is, in poliomyelitis, a complete disorganization of the normal reciprocal innervation because of the simultaneous activation of opposing muscles

Spasticity is therefore a general phenomenon in early stages of infantile paralysis It is quite independent of muscle weakness, and it appears in synergist and antagonist alike It is caused either by increased reflex irritation of hypersensitive muscles (stretch reflex) or is due to lack of inhibition because of the elimination of inhibitory action of the internuncial cells or other cell groups with similar function

3 "Mental alienation"

If this term means suppression of motor function as a higher degree of inhibition, it has a physiological explanation, although exception could be taken to the term Essentially it is a physiological block occurring in the central nervous system and more specifically in the system of synapses which is located all the way from the cortex down to the cord It is altogether transitory and reversible, although for the time being it may appear as a real paralysis

4 Incoordination

This is a phenomenon in which the patient either voluntarily or involuntarily tries to use a stronger muscle for one which is paralyzed Therefore, incoordination is really substitution This concept has found experimental confirmation (Brazier Watkins and Schwab[3]) in the demonstration by action currents of

simultaneous activation of synergists and antagonists, with more or less com plete overthrow of reciprocal innervation

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Lecture II

ON ANTERIOR POLIOMYELITIS THE CLINICAL PATHOLOGY

I ORIENTATION

IN A general infection which involves not only the central nervous system but other systems of the body as well, the clinical picture may be expected to vary within wide limits The present refinement of diagnosis rests more upon the recognition of the preceding systemic signs than upon the interpretation of the localized symptoms of the central nervous system The earlier writers described these symptoms with considerable accuracy even though the pathological back ground was not recognized Jacob Heine (1840 and 1860) interpreted the condition as an infection of the meninges, a misconception which was corrected by Charcot, who recognized the anterior horn lesion as the principal pathologi cal feature

For the presentation of the clinical symptoms referable to the central nervous system we may refer to the classic monograph of R W Lovett[1] The out standing signs of paralysis, rigidity, hyperesthesia and muscle spasm are described by him in a comprehensive manner which requires little addition today

II THE ACUTE PHASE

In the initial stage the systemic signs of fever, rapid pulse, headache, sore throat and gastrointestinal disturbances dominate the picture, and for the first 24 to 48 hours the identity of the condition may still remain obscure Often a short afebrile period of one to four days follows, with subsidence of the general symptoms, and this again is succeeded by a second febrile period (camel hump type of fever curve) with headache, drowsiness, meningeal irritation, stiffness of the neck, tenderness, rigidity, twitching of special muscles and especially a general hyperesthesia Kernig's and Brudzinski's signs are often positive

In a varying number of cases paralysis develops, usually on the third or fourth day after onset. In the 1916 epidemic in New York almost all reported cases were paralyzed in the later epidemic of 1931 only 70 per cent in the 1935 epidemic in Vermont only 50 per cent were paralyzed, in some epidemics the incidence of paralysis has been as low as 5 per cent. This fluctuation in the incidence of paralysis in various epidemics is due to a combination of causes It is certain that in earlier years infantile paralysis was not promptly diagnosed and many abortive and non-paralytic cases escaped attention Then also, there is a definite variation in type and severity of the paralysis among the different epidemics Some are particularly severe and present numerous cases of the bulbar type others are remarkably mild and abound in abortive cases

A. CLINICAL TYPES (*TOOMEY*)

The *abortive type* is confined to general symptoms of fever, headache, stiff ness of the neck, pain along the spine, nausea, vomiting, sore throat, but there is no paralysis, similarly in the *neuritic type* the prevalent symptoms are pain, paresthesias, anesthesias also without paralysis and in the *meningitic type* meningeal signs are prominent So far as paralysis is concerned all these types are abortive and none of them shows any mortality

The most common form is the *spinal type,* involving about 75 per cent of the cases In this type signs of muscular paralysis of variable degree follow the general symptoms The mortality in this group is about 3 per cent. In the *bulbar type* the involvement of the ninth, tenth and eleventh cranial nerves produces dysarthrosis, dysphagia, loss of gag reflexes, inability to expectorate and respiratory paralysis This type which involves 10 to 20 per cent of the cases has a high mortality, over 75 per cent. Paralysis of the *facial nerve* is rare and that of the temporal muscle and masseters, of which we observed one case, is most unusual The so-called *cortical type* is seen rarely except in large epidemics It combines the general preparalytic signs with those of encephalitis, that is, with upper motor neuron lesions, there is no flaccid paralysis and no mortality A survey (D D Young[1]) showed that a number of menstrual irregularities, mostly based on dysfunction of endocrine glands, and psycho-neurosis was frequently observed.

B THE DIAGNOSIS

1 Spinal fluid

In the preparalytic stage the most important diagnostic measure is the spinal puncture In anterior poliomyelitis the spinal fluid count varies from 100 to 700 cells as against 5 to 10 per mm[3] normally[8] It is under pressure equivalent to 150 to 200 mm of mercury and the fluid contains no pus or fibrin webs the reducing substances are normal, 60 to 80 mg per cent. There is no cor relation between the spinal fluid pressure or the amount of globulin in the spinal fluid and the degree of paralysis.[8] The rise in the cell count occurs early but may fall to normal in the second week On the other hand, the protein count is normal first and later rises, to attain its maximum in the third week Conse quently, if one takes a spinal fluid specimen at the end of the second week one may obtain a practically normal cell count since it has already fallen and a practically normal protein count, because its rise has not yet begun One must therefore rely on repeated examination, a persistently normal spinal fluid prac tically excludes poliomyelitis

2. Signs and symptoms

Hyperesthesia and increased tactile sensibility is more diagnostic than the rise in temperature The hyperesthesia ranges from the slightest tenderness to exquisite sensitiveness to touch over all affected muscles This is especially

apparent in the neck and back, less in the muscles of the extremities *Stiffness and rigidity* is characteristic from the very beginning When the patient is trying to bend his neck, he raises his entire trunk because of stiffness of the back muscles (*Caverly s sign*), when he attempts to touch the chin to the chest, he does so by opening his mouth and dropping his chin (*Levinson's sign*) Attempts to extend the knee when the hips are flexed are resisted (*Kernig s sign*) Flex ing the head and neck causes flexion of the knees (*Brudzinski's sign*) Hyper esthesia as well as rigidity are transitory signs and respond readily to symptomatic treatment *Tremor and twitching* are not diagnostic because they occur in other conditions affecting the neuromotor end apparatus, such as progressive muscular atrophy Twitching indicates a slowly progressive degeneration of the anterior horn

Reflexes. The first reflexes to disappear with the advent of paralysis are the abdominal and cremasteric. *Beevor's sign*, i e , the upward or lateral displace ment of the umbilicus on stroking the abdominal wall is seen in asymmetrical paralysis of the abdominal muscles The deep reflexes vary While exaggerated in the beginning of the disease, they finally disappear with the progress of paralysis

The muscle spasm. As already described by Lovett, it is an involuntary in crease of the muscle tone caused by the effect of the virus on the regulatory centers of the spinal cord It is the most common symptom and is found in patients who have been splinted from the onset as well as in patients who have been entirely untreated In the 1946 epidemic in Iowa 62 per cent of the cases exhibited spasm, the hamstrings and quadriceps were affected in 23 per cent, the back, in 27 per cent In the 1948 epidemic 60 per cent showed spasm, the hamstrings preponderating with 53 per cent, the back with 29 per cent and the quadriceps with only 8 per cent (W C Cantrell[1])

Contractures are different from spasm Here the muscle is entirely inert and shows no action current *A true contracture* may occur in a weakened as well as in a normal muscle It is due to shortening of the muscle parenchyma *Contrac tion*, on the other hand, is a voluntary action We have never observed a true contracture in a completely or even in a severely paralyzed muscle They are preventable by proper positional treatment in early stages, and we observed none upon discharge after the acute stage in either the 1946 or the 1948 epidemic In the chronic stage they develop frequently unless special precau tions are taken against their occurrence

The so-called mass movement is a much discussed phenomenon of incoordination which one often sees in patients as they attempt to carry out a cer tain directed motion It is essentially a substitutionary maneuver such as we see in spastic paralysis and is a complex type of motion built up on a complicated motor pattern Essentially, it is an attempt of the patient to change to a combi nation of motion in which the paralyzed muscle can be excluded and a new pattern installed in the motor cortex, in a sense, a detour around a specific motor center which is out of commission

The disease is prevalent in the young although not overwhelmingly so. It varies with the epidemic. In the Iowa 1946 epidemic 61 per cent of the cases were below and 39 per cent above 14 years, with an average age of 10 47 years In 1948, the average age was 12 7 years with only 57 per cent below and 43 per cent above 14 years (W C Cantrell[1])

III THE CHRONIC STAGE OF ANTERIOR POLIOMYELITIS

The two outstanding features of the chronic stage are the residual paralysis and the contracture deformities Of all cases treated during the last two epi demics, these two sequelae were found in 66 per cent of the cases in 1946, and in 53 per cent of the cases in the 1948 Iowa epidemic. Compared with the upper, the lower extremity shows a decided preponderance—47 per cent against 12 per cent in the 1946 and 34 per cent against 16 per cent in the 1948 epidemic (W C Cantrell[1]) The abdominal muscles are frequently involved In severer cases a lumbar lordosis develops in symmetrical paralysis, and scoliosis in asymmetrical paralysis

The paralysis of the musculature of the back leads to paralytic scoliosis which we noted, in spite of early Kenny treatment in 27 per cent of the 1946, and 29 per cent of the 1948 epidemic.

So far as respiratory paralysis is concerned a distinction must be made according to the muscles involved. In paralysis of the intercostals the lesion is in the thoracic cord there is comparatively little respiratory distress, but the breathing is mainly diaphragmatic. In paralysis of the cervical cord the phrenic nerve center located in the third and fourth cervical segments is involved, the diaphragm is paralyzed, and inspiration is embarrassed In bulbar lesion with paralysis of the tenth and other cranial nerves respiration is very difficult, and there is more cyanosis than in either phrenic or intercostal paralysis.

IV SPECIFIC CLINICAL SITUATIONS

A. THE UPPER EXTREMITY

1 *The paralysis of the trapezius* (*Fig* 11)

This causes the shoulder to slump and the acromion to droop forward and downward. The scapula no longer pulled back by the muscle, assumes a more forward position. Normally the trapezius rotates the shoulder blade in cooperation with the serratus anterior thus completing the elevation of the arm. When the trapezius is paralyzed the action of the serratus anterior is impeded not only because it has lost its rotatory assistant, but also because it no longer is held under tension by the lower trapezius fibers This is more marked *in lateral abduction of the arm where the lack of back pull of the scapula* by the trapezius causes the vertebral border to fall away from the spinous processes Consequently abduction of the scapula in the frontal plane is made possible only by the stabilizing action of the rhomboid.

2. The paralysis of the anterior serratus (Fig 12)

The acromion points farther backward and is slightly elevated, because the upward and backward pull of the trapezius is no longer neutralized In anterior elevation of the arm the defect becomes more evident since the principal for ward mover of the shoulder blade is missing Only the pectoralis minor, the mid portion of the pectoralis major and the acromioclavicular portion of the deltoid are left for this movement The vertebral border of the scapula strongly projects backward when the arm is elevated forward (winged scapula)

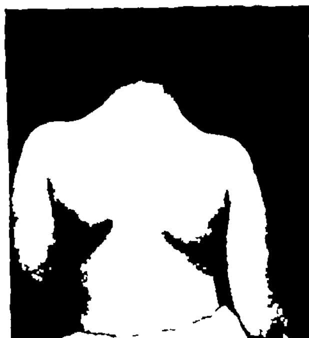


Fig. 11 Paralysis of right trapezius.



Fig. 1 Paralysis of right anterior serratus winged shoulder

3 Paralysis of the deltoid

This muscle is the principal abductor of the arm, its range is limited to the horizontal Its secondary function is the forward and backward movement by the anterior and posterior fibers, as well as outward and inward rotation A substitutionary abductory motion can be carried out with the help of the triceps and biceps when these muscles contract simultaneously and secure the humerus firmly against the glenoid cavity Abduction can then be carried out to a degree by the trapezius and serratus rotating the shoulder blade (Fig 13)

4 Paralysis of the elbow joint

The principal *flexors* are the brachialis and the biceps The brachioradialis is a powerful auxiliary flexor The *extension* of the elbow is supplied by the three heads of the triceps and by the anconeus A slight extensory action is also exerted by the humeral head of the supinator brevis *Supination* is carried out

by the biceps and supinator brevis The supinatory power of the biceps decreases as the supination increases, and the last degree of supination is taken over by the supinator brevis Furtherfore, the four oblique dorsal muscles, namely the abductor longus, extensor brevis, extensor pollicis longus and extensor indicis have some supinatory action.

The principal *pronator* is the pronator radii teres, but with increasing flexion



Fig 13 Deltoid paralysis abduction substituted by rotation of the scapula.



Fig. 14 Flexor paralysis of elbow

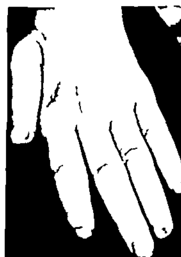


Fig. 15 Thenar palsy

of the elbow muscle loses its pronatory effect For this reason the muscle is paired with the pronator quadratus which is active in all positions of the elbow If all *three flexor* muscles are involved flexion can be substituted only by a forward swing of the whole arm The *extensory loss* may be substituted by gravity *loss of pronation* by abduction and inward rotation of the arm in the shoulder joint The *loss of supination* is much more difficult to supplant, as this would require a forced adduction and outward rotation in the shoulder joint (Fig 14)

5 The paralytic imbalance in the wrist joint

The paralysis of the extensors causes the drop wrist, in which the wrist and

fingers are held in a semiflexed position Paralysis of the long flexors of the fingers associated with paralysis of the intrinsic muscles causes the flat hand with hyperextension in the metacarpophalangeal joint The flat thumb is the result of paralysis of the thenar muscles (Fig 15) It lies in the same plane with the palm and is unable to oppose itself to the fingers

B THE PARALYTIC IMBALANCE OF THE TRUNK

1 The paralysis of the abdominal wall

If this is symmetrical, it causes increased inclination of the pelvis and exaggerated lordosis The abdomen protrudes, and on side bending the flank is not flattened but bulges instead Moreover, the entire respiratory mechanism which depends on the tri-cornered balance between the tension of the diaphragm and of the abdominal wall and the intrathoracic pressure is disturbed We test the paralysis of the abdominal muscles, first, by the *Beevor* sign The umbilicus is pulled toward the non-paralyzed side, upward if the lower portion of the rectus is paralyzed, and sideways in unilateral paralysis of the abdominal muscles

2. The paralysis of the diaphragm

This causes severe respiratory embarrassment because its abolishes the inspiratory depression and the consequent enlargement of the thoracic cavity Normally, the descent of the diaphragm during inspiration causes the abdominal wall to bulge, while during expiration contraction of the abdominal wall together with a relaxation of the diaphragm furnishes the expiratory backstroke. The abdominal cavity acts as a piston driving into the thoracic cavity and emptying it of air The paralysis of the intercostals abolishes the active dilatation and contraction of the chest wall, but respiration is still possible within normal requirements The combined paralysis of the diaphragm and intercostals, however, constitutes a very severe respiratory embarrassment

C THE LOWER EXTREMITY

(See also Vol I, Section A, Lecture IV, On Pathology of the Gait)

1 The hip joint

The *gluteus maximus paralysis* produces a characteristic gait. The patient cannot depend upon the muscle to rotate the pelvis backward, therefore, in the supporting phase of the affected limb the trunk is thrown backward in the hip joint with the anterior muscles of the hip and the iliofemoral ligament acting as a check. In bilateral paralysis the gait is more awkward because this backward thrust of the trunk is repeated with every single step

The weakness of the gluteus medius increases the pelvic oscillations in the frontal plane. In the absence of active abduction the leg hangs in the hip joint and a strong jerk of the trunk to the opposite side is necessary to clear the limb off the ground However, it has been shown that the gluteus medius is not the only stabilizer of the pelvis When the subject is standing on one extremity

with the pelvis level, the force which prevents the pelvis from dropping on the non-supported side is not entirely active muscle pull of the abductors, but to a considerable extent the pelvis is fixed by the passive tension of the fascia lata and the iliotibial tract (V T Inman[7]) In trying to minimize the dropping of the pelvis toward the non supported side the patient, when standing on the side of the paralyzed abductor, shifts the pelvis toward this side so that the line of gravity falls more or less through the hip joint. This naturally minimizes or abolishes any rotatory effect of gravity in respect to this joint.



Fig. 16 Paralysis of quadriceps genu recurvatum.



Fig. 17 Paralytic valgus.



Fig. 18. Paralytic varus.

Fig. 16 Fig. 17 Fig. 18

2. The knee joint

The paralysis of the *quadriceps* (Fig 16) affects the gait much less than one would expect, because as long as the gluteus maximus and soleus are intact they are able to stabilize the knee sufficiently in standing and in ordinary walking It is only in the fast walk or in the run that instability becomes apparent. If the paralysis of the quadriceps is combined with paralysis of the gluteus maximus or of the calf muscles or both then the limb becomes unstable even in slow walking In this case a limp appears, and the steps become shorter The patient tries to prevent the jack knifing of the knee either by throwing his trunk forward so that the line of gravity falls in front of the knee joint axis or by pressing the hand against the knee to replace the missing quadriceps action

Paralysis of the hamstring muscles alone causes little disturbance of the gait so long as the knee is not contracted or deformed If a genu recurvatum develops however the calf muscles become contracted, the foot is in equinus and the gait becomes sloppy (Fig 16)

3 The ankle joint

In *paralytic calcaneus* due to paralysis of the calf muscles, the ankle appears stable in standing and slow walking but in rapid walking and running a marked limp develops There is a complete lack of take-off, and the propulsion is carried on entirely by the heel There is little effect on the oscillation of the pelvis and trunk so long as the legs are approximately equal in length In contrast, in *paralysis of the extensors* the foot drop produces the typical steppage gait The foot is unstable, the take-off is considerably impaired, and the supporting phase is shortened

In the *paralytic valgus* (Fig 17) the gait is impaired because the valgus position of the foot makes it difficult to develop a take-off Propulsion is accomplished only by rotating the foot strongly outward and pressing the big toe firmly to the ground On the other hand in the *paralytic varus* where the weight line passes through the outer border of the foot (Fig 18), practically no real take-off is possible instead, the foot is pushed backward by the flexors of the toes, digging into the ground The ankle is very unstable

D RESIDUAL DEFORMITIES IN CHRONIC POLIOMYELITIS CONTRACTURES (FIG 19)

Contractures arise from the pull of the unopposed muscles, or they are purely postural Postural contractures are frequent Here belong, for instance, the equinus deformity of the foot, the flexion contracture of the knee, the flexion contracture of the hip, the adduction contracture of the arm, the pronation contracture of the elbow and particularly the contractures of the spine The flexion contracture of the hip is the most frequent of all It develops when the patient remains sitting or even lying in bed with his legs drawn up As a final result of the contracture position the muscle undergoes a structural fibrosis and becomes irreversibly shortened



FIG. 19 Paralytic flexion contracture of hip joint

E THE LATE GAIN IN MUSCLE POWER

In the acute and subacute stages the future of the paralyzed muscle is rather unpredictable, and rapid recoveries within the first six months are nothing unusual When the disease has entered the chronic phase, i e , after the period of rapid recovery has passed it will be found that muscles which have been paralyzed for two years without any return in power seldom show any improvement under any kind of treatment. Severely paralyzed muscles not

showing any gain within six months may be considered as definitely lost. We have never been able to see any return of power in muscles classified as 0 or even 2 (poor), when they were paralyzed as long as three months without showing any appreciable recovery In muscles classified as fair or good, im provement in strength may be expected after a much longer time

F THE ULTIMATE SHORTENING OF THE LIMB

The greater the degree of paralysis, the greater is the degree of shortening, the younger the patient is at the onset of the paralysis, the greater also is the amount of shortening On the other hand, walking seems to play little if any part in the degree of ultimate shortening The arrest of length growth occurs during the entire growing period, but it is accelerated during the period of rapid growth On the other hand, premature closure of the epiphyseal cartilage in the paralyzed limb has not been observed (D Ross[4])

V THE DIFFERENTIAL DIAGNOSIS

There are certain clinical features in the acute stage which help in the early diagnosis of poliomyelitis Irrationality or coma is uncommon The temperature is only moderately elevated. Convulsions are rare and occur in infants only Some meningismus may be seen during the stage of acute systemic infection but it is only temporary In general, the symptoms in poliomyelitis are less severe than in purulent meningitis

A THE DYSTROPHIA MYOTONICA

This is a hereditary disease of the central nervous system which affects a number of persons of the same family in different generations and is character ized by muscle weakness and wasting and often cataracts

B THE NEURONITIS OR SO-CALLED GUILLAIN BARRÉ SYNDROME

This is really a polyneuritis which occurs usually in children and young adults It has a slow onset and develops paralysis first of the lower and then of the upper extremities rarely involving the respiratory muscles The course of this disease is afebrile, the paralysis is flaccid, and the deep reflexes are absent. The patient complains of general aches and pains in the calves and back, and the initial symptoms may be mild and pass unobserved. The spinal fluid shows a very high protein content, up to 800 mg per cent, which tends to increase in the acute stage and remain elevated long after recovery In contrast the number of cells is low The weakness most commonly involves the proximal portions of the limb while sensory disturbances are as a rule limited to the distal portions. The motor signs are usually symmetrical and bilateral and are progressive. Most of the cases make a complete recovery The similarity to infantile paralysis lies in the fact that the history is about the same, but there is more pronounced hyperesthesia in neuronitis Exacerbations may develop after some interval which are not seen in poliomyelitis

C THE ENCEPHALITIDES

This is often associated with flaccid paralysis when the disease extends into the cord Spinal fluid changes are absent, however, but the presence of neuro logical signs indicates focal lesions in the brain and there is prolonged fever

D TUBERCULOUS MENINGITIS

This is also characterized by fever, stiff neck and changes in the spinal fluid which may confuse the issue The differential points are the appearance of intracranial pressure, the cerebrate rigidity, the pathological reflexes and finally the findings in the spinal fluid which in tuberculous meningitis show more pleocytosis, greater globulin content but markedly diminished sugar reduction and the characteristic pellicle which appears when the fluid is allowed to stand

E THE TRANSVERSE LESION OF THE CORD

Acute myelitis or Landry s paralysis is differentiated by the ascending tend ency of complete motor and sensory paralysis, the slight rise in temperature and the absence of spinal fluid changes

F ABSCESSES OF THE CORD

These are differentiated by the complete loss of all forms of sensation, with the exception of tactile The bowel and bladder functions are also lost, which is unusual in poliomyelitis The spinal fluid cell count is normal

G THE ALCOHOLIC POLYNEURITIS

This resembles the neuronitis, the diagnosis is made on the gradual progression of the paralysis, accompanied by severe pain and intense sensory disturbances The loss of power is the first symptom and there is muscle twitching The reflexes are depressed but not abolished, and the electric reactions remain un changed

H PSEUDOPARALYSIS

That which we see in Barlow s disease or in congenital syphilis may simulate poliomyelitis The Wassermann reaction of the blood and the spinal fluid establishes the diagnosis in syphilitic lesions, and the concomitant signs of scurvy and the x ray picture identify Barlow's disease

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Lecture III

ON ANTERIOR POLIOMYELITIS CONSERVATIVE TREATMENT

THAT no treatment of any kind can be evaluated without contrasting it with the natural course of the disease is too self-evident to be argued Precisely on that score, it has been asserted that the early treatment of anterior poliomyelitis has little if any effect upon the course or on the degree of residual paralysis (C H Crego[1]) It was pointed out that in cases which were allowed to run their natural cycle, except for supportive treatment, only 10 per cent showed enough residual weakness to require braces or surgery while 72 8 per cent had either no residual weakness or it was so insignificant that no treatment was required (M S Scherman[2]) From the reports one may gain the impression that early treatment is unnecessary Such a conclusion would be justified only if the objective of the treatment were the prevention or the cure of definite and irreversible pathological changes It is well to recognize from the outset that with the exception of the sporadic and very inadequate attempts to control the disease by vaccine and serum therapy *all treatment of infantile paralysis is symptomatic* The question is Does the treatment give symptomatic relief by control of muscle spasm and pain? Does it prevent or correct deformity and disability? Does it relieve cerebral inhibition and revive the normal motor impulses?

I GENERAL TREATMENT

A SEROLOGICAL

The direct attack on the virus infection itself, rational as it is, has been disappointing on the whole The time for the use of *convalescent serum* is the preparalytic stage As a majority of cases do not develop paralysis at all, it is difficult to evaluate the effect of this treatment. Some larger series (A. L Hoyne[3]) seem to indicate that serum therapy does not reduce the death rate and does not prevent paralysis and that there is no merit in the application of serum (J C Geiger R W Burlingame and R C Miller) So far as the use of *vaccine* is concerned it seems that the more active kind (Kolmer) is not without danger while the formalin inactivated vaccine (Park) is said to be ineffective.

B MEDICINAL TREATMENT

1 **Antispasmodics**

Prostigmine This is not a new remedy it has been used for certain other paralytic conditions particularly for myesthenia gravis and was recently popularized for infantile paralysis (H Kabat and N E Knapp[4]) Its physiological action is that it inhibits the function of the cholinesterase an enzyme responsible

for the rapid splitting and inactivation of acetylcholine Prostigmine, in destroying the cholinesterase, permits a greater accumulation of acetylcholine in the synaptic inhibitory centers with the result that the tonus of the skeletal muscles is decreased and with it the proprioceptive reflex irritability It is used as a combination of 1 mg prostigmine bromide with 1/200 gr atropine sulfate hypodermically every three or four hours

The most powerful antispasmodic drug is *curare* Ransohoff, who introduced it into the treatment of poliomyelitis in connection with physiotherapy and particularly muscle stretching, finds that the action currents of the resting muscle can be diminished or obliterated by means of this drug It is given in dosage of 0 9 units per kgm body weight every eight hours for the first 24 hours and 1 5 units subsequently

2. Vitamins and chemotherapy

All reports on the effect of vitamins upon the course and the rate of recovery of the paralysis have been negative The same is true of the chemotherapy The sulfonamides have been tried and found entirely ineffective (Toomey[4]) So far there is no evidence that any of these drugs has any effect upon the metabolism of the host cell upon which the virus depends for its existence

II LOCAL TREATMENT

In a spirit of dissent rather than on the basis of facts it has been claimed that until recently the so-called orthodox methods universally dominated the treatment of infantile paralysis, and that they were founded on the principle of the most rigid and prolonged immobilization This is not entirely correct, because there have been in the past so many departures from the idea of protracted and uninterrupted immobilization, and it has been challenged before in so many quarters that one can hardly call it universally accepted In addition, some newer physiological observations have changed our ideas on complete immobilization Quite to the contrary, it was found that if the muscle passes through its complete range of motion at frequent intervals, recovery is enhanced rather than impeded because of the stimulation of the venous circulation and lymphatic flow

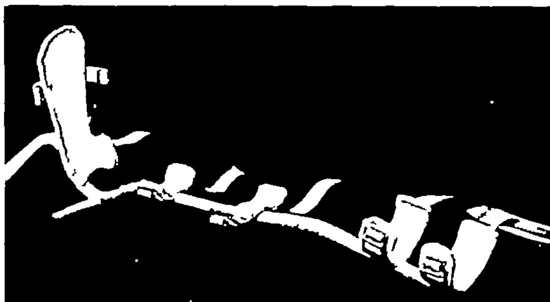
A THE PRINCIPLE OF REST AND RECUMBENCY

What then, is the objective of rest and recumbency? During the periods of hyperesthesia and muscle spasm the patient naturally must be kept in bed, but this period of immobilization should not be unduly protracted and motion should begin at an early date The exception is the threat of paralytic scoliosis where recumbency should be prolonged until all signs of spasticity of the back muscles have disappeared Proper splinting is essential during recumbency to avoid contractures There is no serious objection to the use of splints in order to hold the knees and ankles in the proper position (Fig 20), though it can also be done by proper placing of sandbags

B PHYSIOTHERAPY

When should physiotherapy begin? The application of heat should start immediately, as advised by Lovett and others many years ago

1 The first objective of *heat* is the *control of pain* The method of its application has been greatly improved by the technique developed by Miss Kenny



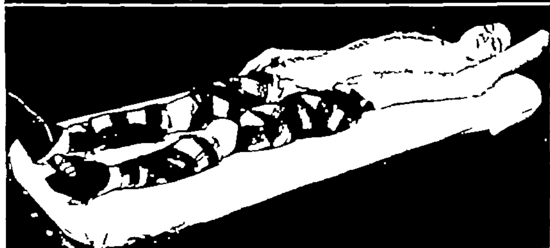


FIG 20 (*top*) Leg splint in recumbency
FIG. 21 (*bottom*) The Kenny pack.

and it is especially effective in controlling muscle spasm Use is made of continuous warm fomentations under a special, carefully devised technique (Fig 21) In case of respiratory embarrassment due to spasm of the intercostals these packs have to be applied very frequently In other instances, the applications made at one or two hour intervals or even less often will suffice In some instances as in the spasm of the intercostal muscles, the relief is striking

2. Stimulation of muscle tone

It is imperative that the tone of the muscles be maintained through the early stages of the disease There are several means of keeping the remaining muscle fibers in good condition The modalities in use are ultraviolet rays, short wave currents, warm water pool and electric stimulation, passive motion and active exercises The ultraviolet radiation has merely a general stimulating effect (G Myley[1]) *Short wave and diathermy* are conveyors of heat which improve the muscle tone by acceleration of the circulation On the other hand, the under water treatment first instituted by C L Lowman is an excellent and direct method to develop muscle tone It facilitates active exercise of weak muscles by eliminating the influence of gravity The warm water pool is particularly useful in the early stages of muscle rehabilitation The procedure can be simplified by using individual so-called Hubbard tanks instead of the expensive warm water pools

There is a question in regard to the value of electrotherapy It improves the circulation by stimulating muscles to contract, which are otherwise unresponsive to voluntary efforts, and it has been demonstrated by physiologists that electric stimulation of certain intensities retards muscle atrophy The interrupted current is preferable to galvanism because of its rhythmic stimulation The surging sinusoidal type has the advantage that it is less uncomfortable than the faradic current

3 Muscle re-education

This is the most important of all physiotherapeutic modalities A central inhibition blocks the transmission of voluntary impulses from the higher centers to certain muscles, these are the muscles which profit most by re-educational methods The details have been carefully worked out under the Kenny technique. The muscle must be taught to resume its function by being carried through its range of motion first passively and then actively This produces proprioceptive stimuli which break through the cerebral inhibition and reestablish the motor pattern in the brain It makes no difference what interpretation is given to the state of the muscle involved, whether we call it mentally alienated or anything else, it is clear that only the weakened and not the completely paralyzed muscle can be expected to respond to re-educational training

The educational program of muscle re-education must be built up by successive steps, each of which represents a locomotor performance more complex than the preceding one The more simple ones are those which are connected with alignment of the body The patient is first taught to sit and to stand, then he proceeds to the function of locomotion, ending up with more intricate and complicated movements of the extremities To work out such a plan in every detail requires all the ingenuity and experience of the trained worker in physical as well as occupational therapy

4 Evaluation of the Kenny method

A heated controversy has arisen among orthopedists regarding the claims and merits of the Kenny method We feel that in one respect at least the method deserves a great deal of credit. It has helped to break a tradition of absolute and prolonged immobilization, which in some quarters had been greatly abused, and which not only disregards established physiological facts, but is also contrary to the old teaching of Lovett. He was a strong advocate of the application of heat and of muscle reeducation So far as the muscle educational part of Kenny's system is concerned it is an excellent and refined method of dissociating substitutionary mass movements by individual muscle training and selective muscle exercises A student of infantile paralysis, Ricardo J Caritat,* points out the fact that muscle reeducation by sytematic exercises was carried out as far back as Ducroquet, before it was advocated by Lovett and his followers. Yet, the fact that others have emphasized the application of heat and the practice of muscle training (Morquio) before should, we believe, not detract from the Kenny method insofar as it represents a definite technical advance

C THE APPLICATION OF BRACES

Should braces be applied at all? The rather peremptory manner in which the question of braces is disposed of in some quarters calls for some remarks. We think that much of this controversy is due to a misapprehension of the purpose of braces They have nothing directly to do with the paralyzed muscle The function of the brace is to support the joint and keep it from jack-knifing Furthermore, its function is to maintain the normal position and the integrity of the joint and to prevent its being distorted We realize perfectly that the muscle is not depending upon the brace for its recovery, nor is the brace indicated because the muscle has lost its function A patient with a paralyzed quadriceps can walk perfectly well without jack knifing the knee, because the tension of the heel cord and of the gluteals is sufficient to stabilize his knee, a brace is certainly not indicated in a case like this Another patient with the same paralysis of the quadriceps has a relaxation of the knee and a tight heel cord and develops a genu recurvatum deformity Such a patient is definitely in need of a brace to *protect a joint which is becoming deformed*

D REGENERATION BY SURGERY UPON THE PERIPHERAL NERVE

Obviously a nerve that has lost all its axis-cylinders is irretrievably lost. The fact is however that in most instances the paralysis is of spotty character and that the destruction of the nuclear columns in the spine occurs in isolated plaques only Consequently most peripheral nerves carry some axis-cylinders which are deprived of their motor horn cells and others which are not. The question arises whether it is possible by some irritative maneuver to produce in the remaining nerve fibers a process of hyperneurotization This means that a number of actually denervated muscle fibers whose motor end plates are no longer con

nected with living anterior horn cells would have to receive a new nerve supply from the axis cylinders of the surviving anterior horn cells This regeneration would have to occur by means of nerve sprouting from the intact axis cylinders and the formation of new neuromotor end plates in the paralyzed muscle fibers Using as a basis this concept of hyperneurotization, Van Harreveld and Billig (also McFarland, Billig, Taylor and Dall') developed the method of the so-called neurotrypsy or nerve crushing

The idea was to crush the peripheral nerve which still contained active axis cylinders in the expectation that there would ensue a regeneration activated by the still existing anterior horn cells and their axis cylinders, and that further more this regeneration would exceed perceptibly the amount of nerve degenera tion caused by the crushing The experiments consisted in pulling out the spinal roots of the sciatic nerve of animals, leaving the fifth lumbar root alone The fifth lumbar root was then crushed on one side, and on the other side it was left intact It was claimed that after the crushed root had regenerated, the power of the muscles supplied by it had increased This was believed to be due to the fact that the nerve regeneration which followed the crushing was so abundant that it actually spread beyond the territory of innervation of the crushed nerve fibers The crucial question in the whole scheme is whether such hyperinnerva tion by a nerve recovering from the effect of crushing actually takes place or not. This point is still under debate Many of the patients we have questioned declared themselves benefited, and the carefully kept records and charts seemed to indicate an increase in strength of muscles rated fair or good From their experimental point of view the physiologists have so far refrained from sanctioning the principle of hyperinnervation Hines[11] and his associates found that the maximum recovery, as measured by muscle weight and muscle tension, which can be expected after crushing a certain nerve is about 85 to 90 per cent of that existing before, and that not only is it not in excess of the preoperative condition but it does not even reach the preoperative level

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Lecture IV

ON ANTERIOR POLIOMYELITIS OPERATIVE TREATMENT

I. ORIENTATION

THE objectives of the operative treatment of residual poliomyelitic deformities and disabilities are 1 The restoration of the normal alignment. 2 The stabilization of joints 3 The restoration of the motor function On the whole, operative indications are conditional on failures of conservative treatment, this means that operations are not indicated unless conservatism fails, but not that the failure of conservatism necessarily indicates operative measures *The problem therefore resolves itself in determining if conservative treatment* has definitely failed and, if so whether operative methods are indispensable One will find that the answer differs not only in the three situations mentioned, i.e., disalignment, instability and motor dysfunction, but also according to the different localizations within these limits We can set up general principles and policies but it takes a detailed situation to formulate the specific indication

1 The normal alignment of the body is destroyed by contractures of the soft tissues which develop during the convalescent stage unless special measures are taken to prevent them. The most common are the flexion contracture of hip and knee the contractures of the ankle joint and the paralytic scoliosis. They are on the whole avoidable and that they appear to be much scarcer today than they were before is due to the recognition of preventive measures and their proper application

The general principle is that contractures call for complete correction by whatever means necessary But the tractability of contracture by conservative means varies greatly with the situation Much of this is due to the difference between the contracture of the short fibered monarticular and the long fibered pluriarticular muscles. The contracture produced by the former is always more rigid and resistant, whereas the pluriarticular muscles allow a much greater degree of stretching. This is illustrated in the difference between contractures of hip and knee In the *former* the contracture is produced by the short uniarticular flexors which give but little and conservative means of stretching are soon exhausted

In the knee on the other hand, the contracture is produced both by the ham strings and the short head of the biceps as well as the gastrocnemius long fibered pluriarticular muscles are paired with short fibered monarticular This is reflected in the manner in which the muscles respond to mechanical correction. Correction by conservative stretching goes much further in the knee than in the hip joint. There is a good reason for pushing conservative measures to

the utmost When the stretched muscle regains its natural length, it is a better physiological unit for future function than any muscle that has been elongated by lengthening its tendon Such lengthening weakens the contractility of the muscle because it destroys the normal relationship between the tendon length and muscle length The point to decide is then, whether the advantage of realignment outweighs the disadvantage of losing contractile power For instance, in the knee joint the contracture of the biceps and in the wrist joint the contracture of the flexor carpi ulnaris may be so rigid that further attempts to straighten the joint by conservative means may lead to dislocation or subluxation rather than to actual straightening of the joint It is here that one must decide on operative measures

2 The second objective is the stabilization of the joint The point is, whether active stabilization by muscle tone can be provided for by a satisfactory rearrangement of the muscular equilibrium, or whether the normal position of the joint can be maintained only by an arthrodesing operation Here one will find great difference in the various joints of the extremities, according to their function The weight bearing joints of the body especially depend mostly upon stability Particularly in the foot and ankle stability is of paramount importance, therefore movement will more often be sacrificed by arthrodesing operations This is in contrast to the joints of the upper extremity in which mobility is the first prerequisite, or in which mobility and stability share equal importance

3 The third objective is the restoration of free and active mobility This applies more to the upper extremity where the dynamic functions prevail while for the lower the main requirement is stability Some operative plans may provide for both mobility and stability, but it is obvious that in the lower extremities where the demand for stability is particularly exacting there will be very few situations in which both static and dynamic functions can be restored to a desirable degree For instance, the elbow joint requires more mobility than stability and an operation restoring mobility may take care of what stability is necessary for satisfactory function In the knee joint and in the ankle joint where the static demands are so much greater, mobilizing operations have a much narrower field of indications

Therefore, when an operative program is decided on upon the grounds that conservative means have become exhausted and that intervention is necessary to overcome the disability, the general principle will be, first, to deal with the disalignment caused by contractures, secondly, to maintain alignment passively by restriction of motion or actively by reestablishing the equilibrium and, thirdly, by procuring mobility by way of muscle or tendon transplantation

II THE SPECIAL OPERATIVE INDICATIONS

A. THE UPPER EXTREMITY

1 The shoulder joint (Loss of Abduction)

a) The Paralysis of the Deltoid

This muscle is the prime abductor in which function it is assisted by the supraspinatus By stabilizing the humerus this muscle prepares the abduction of the shoulder joint which is then taken up by the deltoid. Some degree of abduction can be managed in paralysis of the deltoid by the simultaneous contraction of biceps and triceps which fixes the humerus against the glenoid

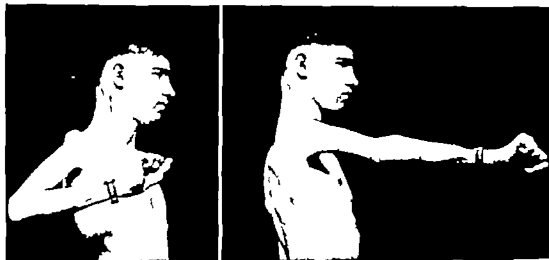


Fig. 22 Paralysis of deltoid fusion of shoulder joint. Case G C. (male) #38-19129 17 months, January 1928 Infantile paralysis four months ago Tendon transplantation of trapezius at age of nine years unsuccessful. Fusion of right shoulder joint at age of 11 years, 1938 good end results. Observation five years after operation.

fossa and enables it to move with the scapula as one mechanical unit. Can the deltoid be substituted by tendon transference? Attempts in this direction have been made for many years More recently L. Mayer[17] has used the trapezius as a transplant by severing the muscle from the scapula and fastening it by means of a fascial strip, serving as a tendon, to the humerus (Fig. 22) It has been our experience that this operation stabilizes the shoulder joint only insofar as it prevents subluxation but there is no useful contraction of the trapezius and the muscle acts more as a tenodesis than as an active muscle. Considering the inadequacy of the transplantation methods, is it practicable to abolish the motion of the scapulohumeral articulation and substitute for it the movement in the acromioclavicular and sternoclavicular joints? The prerequisites are a good trapezius and anterior serratus which rotate the shoulder blade in abductory direction The fact that when the humerus is held fixed in the glenoid some abduction is obtainable by rotation of the scapula suggests that the acromioclavicular and sternoclavicular joints may be adequate to take care of the abduction of the arm after fusion of the shoulder joint.

Our simplified technique of fusion approaches the joint by a circumscribed incision through which ¾ to one inch of the acromial border is resected to provide good access to both glenoid cavity and humeral head and to allow complete denudation The best position after fusion is 50 to 60 degrees abduction for adults and no more than 60 to 70 degrees for children together with 25 to 30 degrees forward flexion (Fig 23) One must avoid too much

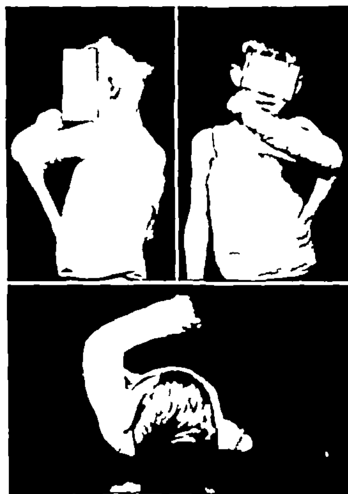


FIG. 23 Arm spica after fusion of shoulder joint.

abduction, otherwise the arm cannot be brought into full adduction after the shoulder has fused (Fig 24) How long one should wait for spontaneous return of the deltoid depends on the degree of paralysis If there is complete paralysis with rapid atrophy (Case B H), there is no object in waiting longer than six months When the muscle is classified as fair, one may wait two years The fusion should not be performed before the age of ten Lately, we have added the resection of the clavicle at the acromioclavicular articulation to increase the abductory range

Our statistics cover 70 cases, in which good solid fusion with abduction to 60 degrees occurred in 74.28 per cent, and fair results where the abduction was 30 to 60 degrees in 18.57 per cent.



Fig. 24 Fusion of shoulder joint in too much abduction. Case B H. (male) #43-1436 12 years, February 1943 Residual deltoid paralysis for six months. Fusion of left shoulder joint in 70 degrees abduction this abduction was too extreme, and patient was unable to bring arm fully to the side. Observation three years.



Fig. 26 Flexor plasty of elbow Case G C.

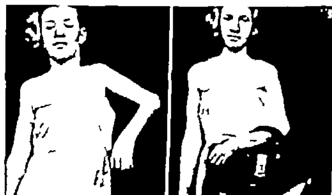


Fig 25 Anterior poliomyelitis complete paralysis of left upper Case M K (female) #41 1486 nine years, February 1941 Poliomyelitis involving entire left upper six months ago all muscles of shoulder joint, elbow and wrist paralyzed, trapezius normal. Fusion of shoulder joint October 1941 Fusion of elbow joint April, 1942 Fusion of wrist September 1942 Active adduction of arm 45 degrees all joints solidly fused, and could use arm as a hook.

In a survey on the end results of the paralyzed shoulder the committee of the American Orthopedic Association[1] comes to the conclusion that arthrodesis of the shoulder is the operation of choice in all cases of infantile paralysis with complete paralysis of the deltoid

b) Paralysis of the Serratus

This prevents close apposition of the shoulder blade to the trunk when the arm is elevated If the abduction is straight sideways in the frontal plane, then the rhomboids hold the scapula tight to the body, and the trapezius is sufficient to rotate the scapula The great difficulty arises when the arm is brought upward in the sagittal plane Then the vertebral border of the scapula stands out strongly (the "winged" scapula) and the elevation in this plane is greatly impaired The muscle acts synergistically with the trapezius in rotating the shoulder blade outward and with the rhomboid in holding the scapula tight to the thoracic wall If serratus palsy is complicated with that of the rhomboids, elevation of the arm is handicapped both in the frontal and sagittal plane The one practical method of substitution for the paralyzed serratus is to insert the sternocostal portion of the pectoralis major into the lower angle of the scapula The pectoralis major tendon can be severed off the crest of the greater tuberosity through an anterior incision and led through the quadrilateral space and then fastened through a posterior incision to the lower angle of the scapula.

2. The elbow joint

a) Contractures

It is not common to see a flexion contracture of the elbow joint develop from anterior poliomyelitis, because the weight of the hanging arm usually prevents it. On the other hand, the pronation contractures of the forearm are frequent due to loss of supinatory muscles, particularly the biceps So far as they resist manipulative correction they can as a rule be relieved by tenotomy of the pronator radii teres Supination contractures are more resistant. Not only is there the contracture of the supinator brevis but the interosseous ligament and the shrunken capsular apparatus also resist correction An osteotomy of the radius and positioning of the forearm in 45 degrees of pronation is recommended by Blount

b) Stabilization of the Elbow

This sometimes becomes part of a larger problem in cases of complete paralysis of the entire extremity which is more often seen in injuries to the brachial plexus than in anterior poliomyelitis There is a solution in case the shoulder girdle muscles are preserved, which consists in fusing all three articulations, shoulder, elbow and wrist, and using the arm as a hook (Fig 25) On the other hand, a common situation in poliomyelitis is the paralysis of the elbow flexors, biceps, brachialis and brachioradialis This condition can be met by our operation of the flexor plasty The condition for this operation is that the muscle

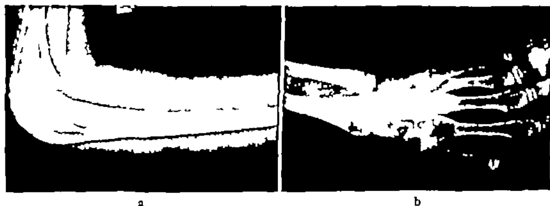
power of the fingers and of the flexors of the wrist is preserved The principle of this operation is to transfer the common head of the flexor carpi radialis the palmaris longus and flexor carpi ulnaris from their common point of origin at the internal epicondyle of the humerus to a point 1½ inches higher up and thus increase the moment of rotation of these muscles relative to the elbow joint (Fig 26) Any pronation contracture existing before operation must be corrected especially since the transposition of the flexors is likely to increase the pronatory deformity

Our statistics show that good results, that is active flexion through a range from 120 to 60 degrees, were obtained in 76 per cent of the cases (A R Smith[11])

3 Paralysis of the wrist

a) Complete Paralysis

This is one of the few situations which call for arthrodesis. It may be part of



a b

Fig. 27 Completely flail arm arthrodesis of elbow and wrist. Case M K. a) Elbow b) Wrist.

a general stabilization scheme which includes the arthrodesis of all three large articulations (Fig 27)

b) Extensor Paralysis

The question arises whether the remaining flexor muscles available for transplantation are sufficient for the stabilization of the wrist as well as for the extension of the fingers This requirement can be met if the extensors of the fingers and wrist alone are involved In this case a tendon transplantation of the flexors carpi radialis and ulnaris to the extensors of fingers and thumb is carried out as in a musculospiral paralysis with wrist drop

If the available muscle material is not sufficient for stabilization of wrist and extension of the fingers for instance if part of the flexors are involved, then the arthrodesis of the wrist must be added to the tendon transplantation.

A third problem is the paralysis of the supinators The lack of active supination is due to absence of the biceps and supinator brevis action, and it leads to a pronation contracture which is very difficult to overcome by a substitutionary

motion in the shoulder The method which we apply and have found very reliable is the transposition of the flexor carpi ulnaris dorsally into the lower end of the radius Through a wide anterior incision this muscle must be thoroughly isolated and led obliquely over the dorsal surface to the distal end of the radius where it is inserted (Fig 28)

Our statistics on the tendon transplantation of the flexors to the extensors



Fig 28 Paralysis of supinators transplantation of flexor carpi ulnaris Case E. A (female) #38-10579 10 years, November 1936 Infantile paralysis involving shoulder muscles biceps, triceps, bilateral inability to supinate left. Flexor plasty left elbow with transposition flexor carpi ulnaris into lower radius December 1938 Flexor plasty right elbow with transplantation flexor carpi ulnaris to finger extensors January 1939 Fusion left shoulder April 194 a) Result of flexor plasty right. b) Result of ulnaris transposition left



show 50 per cent good and 30 per cent fair results Those on arthrodesis, 34 cases, showed 26 or 77 per cent had good results Our figures on the transposition of the flexor carpi ulnaris to supplant the lack of active supination are also favorable Among 16 operations of this kind carried out both for infantile and spastic paralysis good results were obtained in 10 or 66 per cent

4 The thenar palsy

The inability to oppose the thumb to the fingers greatly incapacitates the use of the hand. While it is possible in this situation to substitute extension and

reposition movement by the abductors of the thumb, it is impossible for the thumb to cross the palm and meet the tips of the index and middle fingers The principal reason for this is the absence of the opponens and the adductor of the thumb Our own method consists in the use of the flexor pollicis longus to serve as the opponens of the thumb The principle of the technique is to split the tendon of the long flexor of the thumb into a radial and ulnar half, then to lead the radial half dorsally around to the base of the basal phalanx, so that it now has exactly the direction of the opponens pollicis (Figs 29 30)

Our statistics on flexor plasty of the thumb (J B Davis[8]) are as follows Of 36 cases good and excellent results were obtained in 26, or 72 per cent, fair results in 2, or 5 5 per cent and failures in 8, or 22 per cent. This operation cannot be performed in cases in which the flexor pollicis longus is deficient. Here we advise the method of Bunnell[4] The principle of this method is to substitute the missing opponens by the flexor carpi ulnaris which is elongated by a tendon graft of the palmaris longus and led through a tendon sling fastened around the pisiform bone The elongated tendon is then fastened to the distal end of the metacarpal of the thumb

B THE LOWER EXTREMITY

1 The principle of equalization

The restoration of form involves equalization of leg length A horizontal position of the pelvis is most desirable in order to prevent static scoliosis. When ever the shortening of the leg amounts to more than 1½ inches, an operation for equalization should be considered, either lengthening the affected leg or shortening the sound leg The choice of the method depends on the amount of shortening as well as on the age of the patient

a) The Growth Is Completed

Here again the choice lies between lengthening the paralyzed short leg or shortening of the sound and longer leg

1) Bone lengthening operations. The first bone lengthening operation was suggested by Putti, who after splitting the femur lengthwise by a Z-shape cut used an instrument he called an "osteotone a distending device based upon the turnbuckle principle In this country LeRoy Abbott devised on similar prin ciples an elaborate technique for bone lengthening of the lower leg The indica tion for this operation is a minimum shortening of 1½ inches in young adults with good or fair musculature The operation is of considerable technical diffi culty In order to accomplish the lengthening it is necessary to make an exten sive transverse incision in the vaginal fascia which would otherwise resist the elongation The actual lengthening is accomplished by an extension apparatus which is attached to four pins two above and two below the site of the Z-shaped incision in the bone. The extension must be done gradually, and it takes about

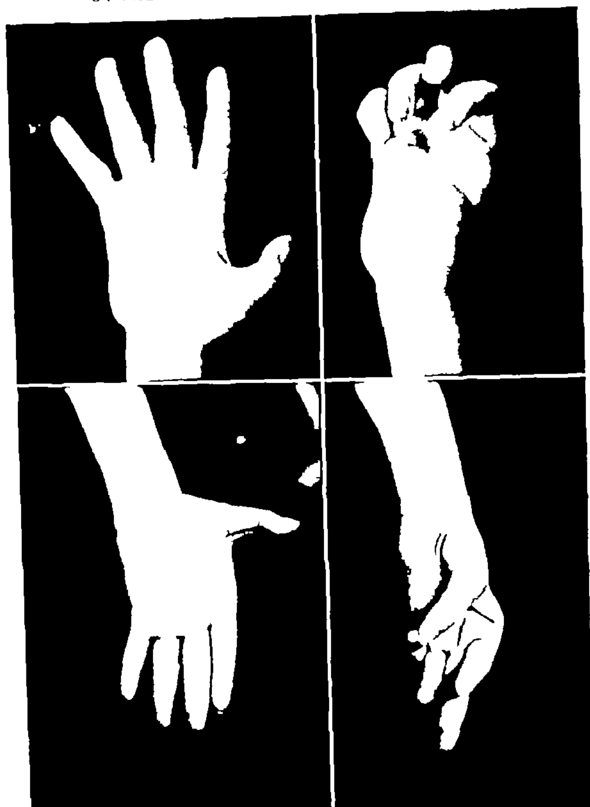


FIG 29 (*top*) Thenar palsy flexor plasty of thumb Case B B (female) #38-2532 five years, August, 1938. Poliomyelitis October 1937 residual thenar palsy paralysis lumbricales, interossei and flexor carpi ulnaris. Flexor plasty of thumb with splitting of flexor pollicis longus good result. Observation two years.

FIG. 30 (*bottom*) Thenar palsy flexor plasty of thumb. Case M K. (female) #M-8.47 seven years, March, 1936 Infantile paralysis nine months ago residual paralysis deltoid, pronators, brachioradialis, flexor carpi radialis, and ulnaris and thenar palsy Flexor plasty of thumb July 1937 fusion of shoulder 1940 Good results from both operations observation seven years.

four weeks in order to accomplish the maximum lengthening which in our cases was about 2 to 2½ inches in the adult.

Our statistics on the lengthening operation (A Bitar[4]) cover 12 cases with an age range of 13 to 25 years In 11 cases tibial lengthening was carried out and in 1, lengthening of the femur The lengthening obtained by the Abbott technique was 1 to 2¾ inches The end result was good in 6 cases or 50 per cent, which means that the shortening of the leg was reduced until there was no more than one inch shortening In 25 per cent the residual shortening was more than one inch, in the remaining 25 per cent there was no improvement whatsoever

2) The bone shortening operations. The shortening of the sound leg is also indicated in cases in which bone growth is completed the operation is much *simpler and less dangerous than the bone lengthening operations* The shortening can be accomplished either by a simple osteotomy as described by White[33] or by resection of a cylindrical piece of bone

b) THE GROWTH IS NOT COMPLETED

Here are also two alternatives either the growth of the sound limb is stopped by a process of epiphyseal arrest, or the growth of the affected bone is accelerated by an operation on the sympathetic system

1) The epiphyseal arrest. This method was first introduced by Phemister, it is suitable only during the latter half of the growing period The physiological age of the patient (in contrast to the chronological) is established on the basis of ossification of the carpal bones according to Todd s table and then the proper time for operation is computed from the growth table of Baldwin Hatcher which gives the expected growth increment for all ages and for the four epiphyseal plates of femur and tibia The upper femoral epiphysis contributes 15 per cent, the lower 40 per cent, the upper tibial 27 per cent and the lower tibial 18 per cent to this growth.

The formula of L R. Straub J C Thompson and P D Wilson[31] is the expectant discrepancy over the expected growth this equals the percentage of growth to be eliminated If it is less than 25 per cent, the operation should be delayed. A more complicated chart based on cumulative x-ray measurements is used by W T Green and Margaret Anderson[16] to predict shortening of tibia and fibula in growing children It seems that the chart devised by these authors gives a more accurate *prediction of the results obtained after epiphyseal* arrest than do the other methods of Baldwin Hatcher, Gill-Abbott[15] or White.[34]

In our own cases we have found the original Baldwin Hatcher method quite satisfactory Our earlier statistics on epiphyseal arrest (A Bitar[4]) gave excellent and good results in 63 5 per cent there was either complete equalization of the legs or at least a substantial decrease of the shortening. A later revision including 100 cases gave 75 per cent good results and 16 per cent poor results with 9 per cent outright failures (Fig 31)

Epiphyseal growth can also be retarded operatively by implantation of metal

or staples across the epiphyseal line (S L Haas,[11] C I Blount)

2) An increase of length growth can be obtained by operation upon the sympathetic nervous system, a method first introduced by R I Harris,[13] in 1930 The physiological principle is that there is a prolonged increase in the blood supply at the growing epiphyseal plate which enhances the growth, provided that the effect of the sympathectomy on the blood supply is per

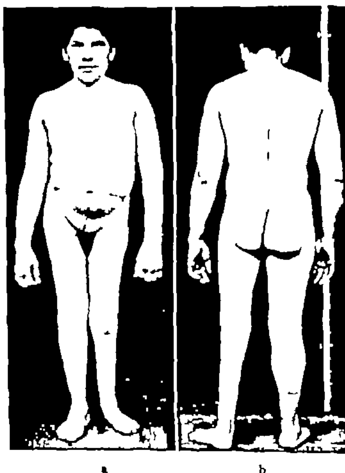


Fig. 31 Equalization epiphyseal arrest right. Case C. H (male) #38-15754 two years, January 19_5 Infantile paralysis at age of one year Considerable shortening left limb with static scoliosis at age of eight years, in 1933 Shortening of 1½ inches at age of 12 years in 1937 Epiphyseal arrest upper and lower tibia. Shortening amounted to ⅛ inch in 1941 Observation four years after operation. a) 1937 b) 1941

a b

manent. The indication is in the growing child with paralysis of moderate degree limited to one extremity It should be performed early, if possible at the age of six.

3) X ray radiation and epiphyseal growth The possibility of retarding epiphyseal growth by radiation was investigated by J A Reidy, J R Lingley, E A Gall and J S Barr,[14] who found that the epiphyseal cartilage was affected by the x ray However, there is the danger of unequal growth of the epiphyses which may lead to considerable deformity

2. Contracture disalignments

a) Flexion Contracture of the Hip

It has been explained that the muscles primarily responsible for the flexion contractures are the monarticular muscles and that these contractures readily



a



b

become rigid and uncontrollable by conservative means When the limit of the conservative treatment has been reached tenotomies and myotomies are called for but even these procedures have their restrictions. The Soutter or Campbell operation is simply a method of stripping the contractured structures from the os ilii using the anterior approach of Smith Peterson

The more immediate results of our own 102 cases (J B Davis[12]) were satisfactory correction in 77 6 per cent of the cases It seems however, that these results are not lasting and that recurrences of flexion contractures were frequent after the Soutter or Campbell operation It is for this reason we are more and more inclined to do subtrochanteric osteotomy either alone or combined with the soft tissue operation to complete the correction (Fig 32)

FIG. 32 Flexion contracture of hip subtrochanteric osteotomy Case L K. (male) #41 4383 14 years, April 1941 Infantile paralysis eight months before flexion contracture in both hips, 90 degrees on right. Conservative treatment failed. Subtrochanteric osteotomy right observation six years. a) Before. b) After

3 The paralytic dislocation of the hip

As soon as the hip dislocates, the patient is no longer capable of bearing weight The alternatives are either to reduce the hip into the socket and secure its position by a shelf or to arthrodese the joint The decision depends upon the state of the abductors If they are in fair condition, it is possible that reduction and construction of a shelf is sufficient If the abductors are severely paralyzed, one can expect very little weight bearing ability even after reduction, and the patient retains the side lurch characteristic of the gluteus medius paralysis In this case stability can be obtained only by arthrodesis The operation should preferably be a combination of the intra with the extra articular method

The position of the ankylosed hip must be carefully calculated, it should be in 25 degrees flexion, no adduction and neutral rotation If the hip is com pletely dislocated and cannot be reduced, the fusion must be done "in situ" which is very difficult to obtain In case there is some abductory power left, it is preferable to perform the osteotomy of the "low" Schanz type so that the weight stress can be deflected in the direction of the shaft

4 The paralytic deformities of the knee joint

a) CONTRACTURES

The flexion contracture of the knee may become very resistant due to the structural shortening of the soft tissues While milder cases will yield to gradual correction under cast or turnbuckle treatment, established contractures may require operative interference in the following sequence 1) *Tenotomy* of the hamstring muscles, 2) *Capsulotomy* of the knee joint capsule, 3) *Osteotomy* of the lower end of the femur

The tight hamstrings are the first obstacle to correction The next is the contracted posterior capsule, which requires the capsulotomy in addition to the tenotomy of the hamstrings Following the technique of P D Wilson[14] the iliotibial band is first sectioned transversely, the external popliteal nerve is freed and retracted and the biceps tendon severed A similar exposure is made from the medial side The operation practically isolates and exposes the posterior border of the articular margin of the condyles of the femur, and by sharp dissection the capsular attachment is stripped off the posterior aspect of the femur

In our series of 12 cases 58 per cent had good and 33 per cent fair results, the standards were for "good," less than 10 degrees lack of full correction, and for 'fair," 10 to 15 degrees.

However, it is important in the paralytic knee to obtain complete correction, since any remaining flexion in the absence of a good quadriceps is likely to lead to jack knifing and instability For this reason one will have to resort in many cases to a final supracondylar osteotomy The most lasting correction is obtained after the growing period, where the action of the soleus imparts a good

backward thrust to the tibia The varus or valgus deformity of the paralytic knee does not immediately cause jack knifing, but it makes for instability of the joint, especially in the growing child in whom the deformity is almost sure to increase unless corrected

b) The Stabilizing and Mobilizing Procedures for the Paralytic Knee Joint

Situation I If the knee is straight and is not deformed and there is paralysis of the quadriceps but the calf muscles and the gluteals are in good condition, any substitution of the quadriceps is unnecessary, because the gluteals and calf muscles take care of the stabilization of the knee. The patient can walk and even run with the amount of stabilization which the knee receives from the above-mentioned muscle groups



Fig. 33 Genu recurvatum.

Situation II The quadriceps is paralyzed and the knee is straight, the triceps muscle is good but the gluteals are poor In this case walking is still possible without support, but the patient assumes the characteristic backward lurch of the paralyzed gluteus maximus He can walk without a brace, although his steps will be short, and he is unable to run Here the transplantation of the tensor fasciae alone is sufficient.

Situation III If there is paralysis of the quadriceps and also of both gluteals and gastrosoleus and the knee is straight, the patient's walk is greatly handi capped The steps are short, the gait is awkward and laborious, and the stabilization of the knee joint by transplantation of the hamstrings, if they are present and available, is very desirable In this case both inner and outer hamstrings should be transplanted into the patella.

Situation IV If the knee is flail and there is paralysis of both extensors and flexors, a considerable strain is placed upon the joint and a genu recur vatum often results (*Fig 33*) If triceps and gluteals are functioning, no opera tion is necessary unless the recurvatum deformity is excessive If in addition to the flail knee however, there is paralysis of the calf muscles, the patient is still able to walk as long as the knee is straight, although he has no deploy and the leg is used as a peg On the other hand if the knee is flail but straight and if both gluteals and calf muscles are poor the decision then lies between the complete stabilization of both ankle and knee joints, on the one hand, and the brace on the other *In this case the decision can be left to the patient* since the operation should not be performed before the age of sixteen Should operative stabilization be preferred to wearing a brace the method of choice is that of

Hibbs, and the position of the joint should be 15 to 20 degrees in children and 25 to 30 degrees in adults, according to occupation

So far as the genu recurvatum is concerned (Fig 33) a number of procedures have been devised to avoid the stiffening operation Mayer s knee check operation[17] fuses the patella to the tibia as a check against hyperextension The modification of Milgram provides for the establishment of a patellar check entirely extra articularly Another suggestion by C H Heyman[11] is based upon the construction of the check ligaments on the grounds that hyperextension of the knee can be prevented by competent anterior cruciate and anterior collateral ligaments Also a method for the genu recurvatum is that proposed by C E Irwin[13] It consists in an osteotomy of the tibia and fibula at the level of the tibial tubercle The principle of the operation is that by suspending the extremity from an overhead Kirschner bow the slope of the tibia is changed from downward and forward to downward and backward

We have tried the check operation of Mayer on several cases, but we have never become convinced that the results are permanent

5 The ankle and tarsal joints

There are specific indications for each of the many clinical situations, but in general, the arthrodesing methods prevail because the static functions make a high demand on the stability of this joint

Situation I. *The Isolated Paralysis of the Tibialis Anticus* This is the only situation which lends itself to tendon transplantation without any additional arthrodesing operation The method of choice is the one devised by K I Biesalski and L Mayer[3] The principle of this operation is to transfer the peroneus longus through the sheath of the tibialis anticus and anchor it to the point of insertion of this muscle[23]

Situation II *The Isolated Paralysis of the Peronei* This leads to a paralytic varus deformity Some believe that this situation also lends itself to tendon transplantation alone The method, also described by Biesalski and Mayer, consists in transplanting the tibialis anticus through the sheath of the extensor longus of the toes to the peroneal tubercle of the fifth toe There is good reason to doubt the adequacy of this procedure in the case of a paralytic varus, unless it is combined with the arthrodesis of the subastragalar joint

Our statistics on the tendon transplantation of the paralytic foot without stabilization were disappointing (W Cooper[7]) In 47 cases good results were obtained only in 14, or 30.5 per cent, fair results in 9, or 18 7 per cent and poor results in 24, or 50 per cent. Most of this poor showing is due to the inclusion of paralytic varus in this series

Situation III. *Both Tibialis Anticus and Posticus Are Paralysed* Tendon transplantation alone is insufficient. This situation calls for arthrodesing operations The method of choice is the so-called triple arthrodesis (Ryerson[30]) in which both the subastragalar and the midtarsal joint are fused *If the valgus*

deformity is accentuated, the arthrodesis can be combined advantageously with the transplantation of the peroneus longus to the tibialis anticus according to the technique of Biesalski and Mayer Our statistics bear out the advantage of such a combination Among 48 operations of this type there were 71 1 per cent good and 18 8 per cent fair results

An older operation serving the same purpose is that of M Hoke.[22] It is essentially a triple arthrodesis in which the scaphoid is removed with resection and reshaping of the head and neck of the astragalus. This combines the stabilization with correction of the form of the foot. Our statistics on the Hoke type of triple arthrodesis (K. F Pelka[39]) showed 88 per cent good results in 91 cases The principal point is that the operation provides a stable ankle both in the subastragalar and in the midtarsal joint as well as the correction of any deformity which might have developed by adaptive changes of the bone

Situation IV *Both Lateral Muscles (Tibiales and Peronei) and the Extensors of the Toes Are Paralyzed Paralytic Drop Foot* The triple arthrodesis is not sufficient. While it stabilizes lateral motion, it does not control the foot drop Campbell[6] devised a posterior bone block which would prevent the foot from going into plantar flexion The block is formed by a mass of cancellous bone piled upon the posterior process of the os calcis, or by a cortical tibial graft fastened to the posterior tibia and pointing to the os calcis (Thornton) Unfortunately these grafts wear off, and the initially good result is likely to deteriorate.

In our series of 33 cases of the Campbell bone block operation combined with the triple arthrodesis (O M Jones[24]) good results (not over 90 to 105 degrees equinus and painless weight-bearing) were obtained only in one-third of the cases, and 66 per cent failed because the bone block ultimately wore off It seems that the solution of the problem of foot drop combined with lateral instability can be solved by osteoplastic methods within the *tarsus itself* A very ingenious procedure is that of Lambrinudi It is based upon the idea of giving the astragalus the maximum plantar flexion and the os calcis the maximum dorsiflexion, by excising a broad wedge with distal base from the head and neck of the astragalus



FIG. 34 Paralytic foot drop Lambrinudi. Case C. A. (female) #45-1344 nine years, May 1948 Paralysis of lateral muscles as well as of extensors. Lambrinudi operation good stability with correction of foot drop

When the two bones the astragalus and os calcis the former in plantar flexion and the latter in dorsiflexion, are now tightly adapted to each other, no further plantar flexion is possible. In addition the pointed head of the astragalus is lodged into a niche made in the scaphoid. The lateral instability of the foot is

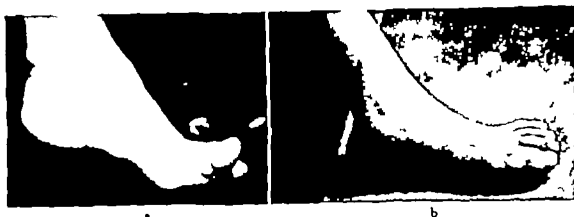


FIG. 35 Paralytic clubfoot with retraction of big toe Case A. A (male) #40-1405 17 years February 1940 Infantile paralysis at age of one year residual paralytic equinus with retraction of big toe Triple arthrodesis plantar stripping and Sherman operation of big toe good correction Observation two years a) Before b) After

taken care of by the added triple arthrodesis Lambrinudi himself (Hart[24]) postulated that the operation has its best effect if there is a good gastrocnemius muscle, but we do not find this absolutely necessary (Fig 34)

Situation V *The Paralytic Drop Foot Combined with Retraction of the Toes* In this case the triple arthrodesis may be combined with plantar stripping and the Sherman operation in which the long extensor of the toes is implanted into the neck of the metatarsals (Fig 35)

Situation VI. *Paralysis of the Triceps With or Without Lateral Imbalance* In this situation the gait is still steady, but it is greatly handicapped by the inability of the patient to deploy the foot He becomes a heel walker, and due to this anteroposterior imbalance running becomes impossible In this type of deformity it is most desirable to displace the foot backward against the tibia. There are two ways to accomplish this The horizontal transverse section of Davis[13] and the astragalectomy with backward displacement of the foot of

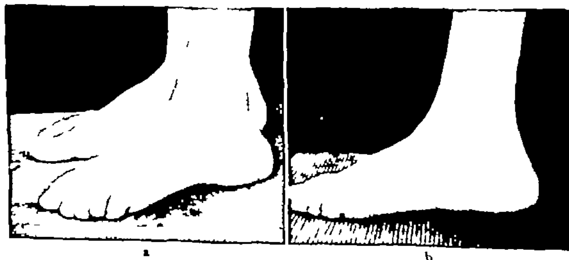


FIG 36 Paralytic calcaneovalgus Whitman operation. Case M. B (male) #38-15158, six years, October 1935 Infantile paralysis at age of two years residual calcaneovalgus. Astragalectomy June 1939 at age of nine years a) Before. b) After.

Whitman[27] The horizontal transverse section, as the name implies, separates completely the astragalus from the os calcis and by removal of the scaphoid and part of the head of the astragalus allows the latter bone to be slid forward on the os calcis Naughton Dunn's method[14] embodies a similar principle

The operation of Whitman adds to the old astragalectomy the essential point of backward displacement of the foot (Fig 36) In addition, the peronei tendons if preserved can be used to supplant the missing gastrocnemius by implanting them into the posterior process of the os calcis

Our statistics on the astragalectomy with backward displacement in cases of paralytic calcaneocavus (J B Davis[10]) showed 88 per cent good results in cases of calcaneo valgus However, in the paralytic talipes equinovarus the results are much poorer 54 per cent good. The condition most suitable, for this operation is the paralytic calcaneocavus

Situation VII *The Drop Foot Without Lateral Instability* In a previous paragraph we have discussed the situation where a drop foot is associated with lateral instability of the foot so that both an arthrodesing operation and a check operation must be performed In this respect we felt that the Lambrinudi operation is the method of choice. A procedure more radical than the posterior block but more certain in its effect is the astragalotibial arthrodesis recommended lately by D B King[22] It entails the sacrifice of the ankle joint and has two serious objections. First there is a considerable strain on the transverse arch because of the lack of buffer like motion in the astragalotibial joint, secondly it entails a considerable backward thrust upon the tibia which puts a strain on the knee joint It is not so well tolerated by adult patients.

Our statistics on arthrodesis of the ankle joint alone (J B Davis[11]) covers 10 cases only three of which however, were poliomyelitis Of the 10, five resulted in fusion and five failed to fuse. The result in the fused cases was good.

Situation VIII. Finally, there are cases in which the foot is perfectly flail and there is no deformity except on weight bearing The knee has good flexors, but the foot is otherwise flail These are cases in which the best procedure is the so-called pan-astragalar arthrodesis, that is, arthrodesis of all articulations of the astragalus It was first introduced by the Belgian surgeon Lorthioir,[26] and later was taken up by Craing,[8] Albee[1] and the writer[33] The operation fuses the astragalus to all the bones with which it articulates, the tibia, the os calcis and the scaphiod in addition to an arthrodesis between the cuboid and the os calcis. The indication is limited to cases of flail feet which show no deformity and which still have good hamstrings The latter point is important because the same backward thrust on the tibia develops which we have described in the tibioastragalar arthrodesis

Our statistics on pan-astragalar arthrodesis (W R. Hamsa[19]) covering 85 cases showed good results with a firm ankle and no disability in 74 per cent of the cases

Summary On the whole we find that for stabilization of the lateral balance of the foot the triple arthrodesis is the most efficient method For correction of

the foot drop the Lambrinudi operation seems to be the best solution The drastic method of sacrificing the entire ankle joint is not justified except in the cases of extreme foot drop In the completely flail foot without deformity and with good hamstrings the pan-astragalar arthrodesis is the proper procedure The calcaneocavus or valgus is best taken care of by Whitman's operation or by the horizontal transverse section of Davis A partial stabilization by tendon transplantation alone without bone operations almost invariably results in failure

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Lecture V

ON THE PARALYTIC SCOLIOSIS

I. PATHOMECHANICS

AN INTRINSIC passive equilibrium is established in the normal spine by the spinal ligaments and the intervertebral disc. The primary cause of idiopathic scoliosis is the loss of this equilibrium. In the paralytic scoliosis there is a loss of the dynamic or active equilibrium which is provided by the muscles controlling spinal movement. Curvatures of the spine are seen in other conditions in which the normal muscular balance of the back muscles is lost. The so-called empyematic scoliosis *is caused by the pull and traction of the shrinking fibrous* masses in the pleural cavity. What is termed sciatic scoliosis is produced by contraction of certain portions of the back musculature in response to a sensory stimulus. These deformities are not true scolioses but only lateral deviations. *They lack the rotatory element which is an essential part of the deformity.* If muscular contractures can only produce lateral deviation, and not rotation as well, they cannot be considered an adequate cause of scoliosis. It is obvious, therefore, that in the paralytic scoliosis one must look for another factor if one *is to explain both the translatory and the rotary component of the deformity.* The simple muscular imbalance produces only the inclinatory deformity, in which one spinal section is inclined against the other, within the limits of normal ranges of motion.

It is true that the paralytic scoliosis often starts as a purely inclinatory deformity but either from the start or in the course of development the rotatory element and the translatory shift always appear, and the spine penetrates into the convexity of the thoracic cage, the same as in idiopathic scoliosis. This *additional pathological factor must be one which destroys the passive* equilibrium of the spinal column. Since the latter depends upon the integrity of the ligamentous structures and of the disc, it is logical to assume that pathological changes involving these structures and especially the disc are responsible for *the rotatory and translatory displacement of the vertebrae (Farkas[3])*. They may be superimposed to a previous muscular imbalance, but they certainly must be operative at some time to produce the full picture of paralytic scoliosis.

Muscular imbalance alone does not explain how the scoliotic spine can assume *degrees of deformation which exceed the physiological ranges* of motion between the vertebrae. The translatory shift of the vertebrae and the excessive rotation and inclination can only come about by structural changes of the disc and the ligamentous structures. Farkas[3] believes that commensurate *changes can be seen in the x-ray picture. The degenerated disc* becomes narrow and soft, losing its firm connection with the vertebral bodies which enables them to undergo a translatory as well as a rotatory displacement.

Can a true scoliosis be produced by experimentally created muscle imbalance? A curvature of the spine was obtained in animals by Schwartzmann and Miles[1] after unilateral removal of the superficial or deep back muscles in rats and mice Many other investigators were able to produce lateral deviations by similar experiments It is doubtful that these experimental deviations were true curvatures, since they showed no pathological degree of rotation While muscular imbalance may be the primary factor, it appears from all experimental evidence that it alone is not capable of producing a true paralytic scoliosis and that another intrinsic factor is required

In many cases the first stage of the curvature is of the inclinatory type, that is, one section of the spine is inclined against the other For some time this inclination remains within the normal ranges of motion, and up to this point the condition can be considered as merely postural Other cases show a rotatory deformity from the start, even before the lateral inclination occurs This rotatory element is at first also within the normal physiological ranges of motion between the vertebrae In the normal spine certain physiological movements impart inclinatory and rotatory stresses, which keep the spine movable and limber These movements are the oscillations of the pelvis, the rotation of the thorax and shoulders during the gait, and the respiratory movements of the thorax In the paralytic these movements are asymmetrical, and this asymmetry is a contributory factor in paralytic scoliosis Scoliosis never develops in complete symmetrical paralysis of either the back muscles or the abdominal wall

The most powerful of these factors is the rotation effect of the pelvis during the gait Asymmetrical oscillations definitely affect the *lumbar spine* Similarly, the asymmetrical paralysis of the upper extremity and of the muscles of the shoulder girdle has an effect upon the *upper thoracic column* The uneven respiratory movement of the thorax in unilateral involvement of the respiratory muscles together with the asymmetrical paralysis of the abdominal musculature has a deforming effect on *the dorsal spine* A curve from the sixth dorsal to the third lumbar develops from a combination of abdominal and lower extremity paralysis A lumbar curve from the tenth dorsal to the fourth lumbar is seen in paralysis of the latissimus, abdominal muscles, iliopsoas and the abductors of the hip joint

II THE CLINICAL PATHOLOGY

A STATISTICS

Scoliosis is a frequent residual deformity of anterior poliomyelitis in cases in which the upper extremities and the muscles of the trunk are involved It constitutes about 20 per cent of all types of lateral curvature How soon after the attack the deformity develops depends more upon the degree of paralysis than the age of the patient We found no preponderance of age in our series where the ages ranged from three to eighteen years The time elapsing from the onset

of paralysis to the appearance of the scoliosis also varied in wide limits, f one month to fifteen years (Armalavage[1])

B TYPES

1 The right thoracic curve



FIG. 37 Long right total paralytic scoliosis. Case L. B (female) 10 years, October 1943 Duration of paralysis three years never stood up or walked. Sitting curve slowly progressing no counter curve.

This is the most frequent common is the *right total curve* (37) The majority of curves are in lower dorsal region, extending bey the dorsolumbar junction, with apex at the ninth tenth or eleve vertebrae

2. High dorsal curves

These develop from paralysis of muscles of the shoulder girdle of from weakness of the convex s deltoid which makes side bend necessary to abduct the arm (I 38)

3 The lumbar scoliosis

This is more often left than rig the same as in the idiopathic ty According to P C Colonna a F Vom Saal,[2] the unilateral paraly of the trunk causes a lateral deviati and rotation of the spine with t *concavity* toward the stronger si except if the psoas is involved in whi case the *convexity* is toward t stronger side

4 The fixed paralytic pelvic obliquity

The imbalance of the abdominal muscles and of the muscles of the bac produces disalignment between thorax and pelvis This results in a pelv

FIG. 38 (*top*) High dorsal paralytic scoliosis with deltoid paralysis. Case J R. (female) #40-1: 13 years, January 1940. Poliomyelitis at age of three years left high dorsal curve. Treated co servatively by development of right dorsolumbar countercurve. *left* Age 13 years. *right* Age years.

FIG. 39 (*bottom*) Paralytic scoliosis persistent extensibility Case L. S. (female) #39-7409 : years, September 1939 Infantile paralysis at age of 2½ years in bed for five years. Residual rig total curve still extremely extensible. No countercurves marked overhang untreated. *left* Supine. *right* Standing.



FIG. 38 top. FIG. 39 bottom. (*legend on opposite page*)

obliquity which becomes fixed by muscular contractures In addition, unilateral paralysis of the pelvifemoral muscle will destroy the normal balance between pelvis and lower extremities The pelvic obliquity therefore results from both pelvithoracic and pelvifemoral imbalance (L Mayer[4]) In 66 cases of paralytic lumbar scoliosis examined we found 20 cases of fixed pelvic obliquity (Armalavage[1])

III. DIAGNOSIS AND COURSE

The natural course of the paralytic scoliosis is unpredictable. It is certain that it progresses rapidly and soon gets out of control It does not seem to come to a stop with completion of growth as the idiopathic scoliosis does Al though the progress may be slowed down, the abnormal extensibility of the spine often persists into middle age, which means that progression of the curve is still possible (Fig 39)

In general it can be stated that the curves which develop under the in fluence of pelvic rotation have a better prognosis than the thoracic curves. Once the fixed pelvic obliquity is overcome by operation or otherwise, the con trol of the curve itself through derotating and corrective maneuvers of the pelvis is much easier than in the thoracic spine.

The spontaneous arrest of the paralytic curve depends as much upon the condition of the non-contractile soft structures of the spinal column, i.e., the discs and ligaments, as it does upon the control of the muscular imbalance. These structures have to regain their normal physical properties so as to resist further displacement of the vertebrae both in the translatory and rotatory sense Unfortunately, this recovery of the non-contractile structures seems to occur late if at all allowing the scoliosis to progress rapidly

IV THE TREATMENT

A PROPHYLAXIS

Prevention of scoliotic deformities depends upon restoration of muscle bal ance in the early stages of poliomyelitis by an alternating program of active exercises and rest. Recumbency should be longer than usual, and the back muscles should be protected from fatigue by bed rest and intermittent, not continuous brace support. In addition certain supportive measures are

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Fig 40 (*top*) Total right dorsal paralytic curve conservative treatment. Case H. L. (male) six years, October 1939 Infantile paralysis two years ago spinal curve noticed 1 month later X-ray long right thoracic curve no rotation. Conservative treatment no progression of curve. *left* X-ray 1937 *right* X ray 1942

Fig. 41 (*bottom*) Paralytic left lumbar curve conservative treatment. Case L. S (female) #44540, eight years, October 1940 Infantile paralysis three years ago involving right upper and right abdominals Slight left lumbar curve stationary Observation 3½ years. *left* October 1940. *right* March, 1944

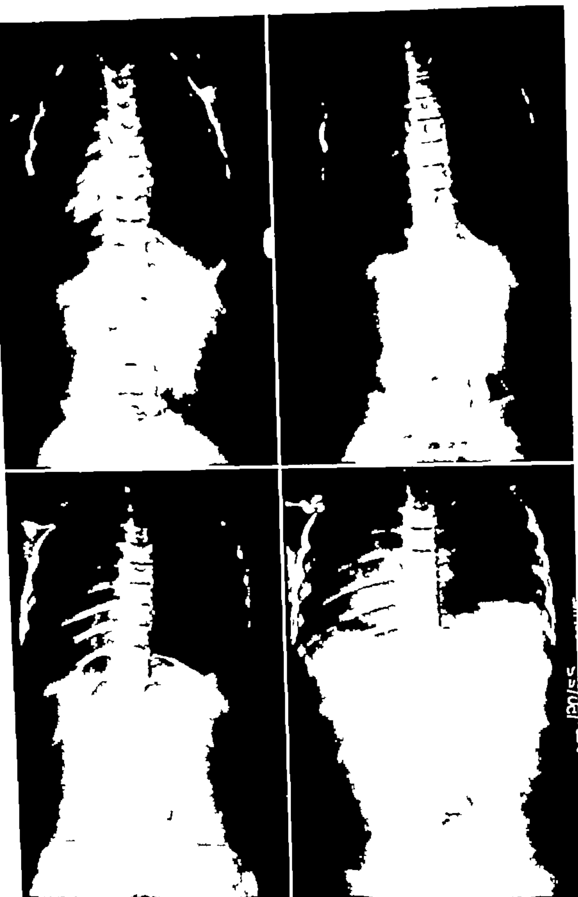


FIG. 40 top FIG. 41 bottom. *(legend on opposite page)*

necessary to promote recovery of all structures of spine and thorax, not only the contractile ones Aside from a proper general regime, a good deal can be expected from systematic exercises to increase the vital capacity Colonna and Vom Saal,[*] using a respirometer, found that cases with curves in the thoracic region have a greatly lowered vital capacity It is evident that with the improvement of the respiratory excursions not only the muscles but also the *other soft structures of the spinal column are benefited, not to speak of the* general constitutional effect.

B CONSERVATIVE

The conservative treatment consists in symmetrical development of back and abdominal muscles by daily exercises, between treatment periods the *patient alternatingly is given bed rest, preferably in traction, and ambulation* under the protection of an adequate support (Fig 40) The relative amount of bed rest and ambulation is determined by the progress in the active control of the spine. There are only a few cases in which conservative treatment is adequate. One group is the mild case of right total curves with little rotation. Another type is the left lumbar curve which can be controlled by conservative means, provided there is no residual pelvic obliquity (Fig 41) A third type is the purely static curve due to a short leg after the pelvitrochanteric obliquity has been corrected (Fig 42)

C OPERATIVE

All cases except the groups mentioned above must be subjected to operative fusion if the progress is to be halted While the operation should not be unduly delayed there are some important preparatory steps which should precede the fusion

1 Restoration of body alignment by compensatory curves

In this respect one can go farther than under conservative treatment. As long as the operative fusion is in the program, one does not have to curtail the mobilizing effort as one has to do under a conservative regime Under conservative management it is essential that the spinal column should not be too limber to maintain its balance actively by muscular effort. In the case of a prospective

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FIG. 42 (*top*) Short leg left lumbar curve in paralytic. Case B S (female) #38-19992, 18 years, June, 1938 Acute polio age 15 months, involving left lower shortening of 2½ inches, with left lumbar scoliosis. Horizontalization of pelvis by extension shoe scoliosis under control with slight increase. *left* Age 18 years *right* Age 23 years.

FIG. 43 (*bottom*) Severe paralytic scoliosis fusion. Case M. S. (female) #38-19200, 16 years, September 1936 Infantile paralysis at nine years paralysis of abdominals. Spinal curvature noted eight months later severe dorsolumbar left curve. Correction by Risser Jacket, followed by fusion from sixth dorsal to sacrum. In spite of incomplete compensatory curves, fusion held; observation 3 ½ years *left* Before fusion 1936. *right* 2½ years later



FIG. 42 top FIG. 43 bottom. *(legend on opposite page)*

fusion this restriction does not apply and one may go much farther in developing compensatory curves by corrective casts The Risser cast is the best method to obtain passive compensation.

2. The horizontalization of the pelvis

In unilateral paralysis of the oblique abdominal muscles and quadratus lumborum there develops a lumbothoracic scoliosis convex toward the paralyzed side, and the pelvis is tilted downward on the convex side. However, if the quadratus is not paralyzed, the same lumbothoracic scoliosis develops, but there is no downward tilt of the pelvis and no fixed obliquity (L Mayer[4]) In some cases the pelvic obliquity can be corrected by traction In most cases, however, no permanent correction can be obtained by these conservative means, and one has *to resort to such operations as the fascial strip suspension in order* to secure the horizontality of the pelvis.

Mayer[4] found that in 38 cases with 46 operations the fascia healed securely in all of them, and recently Wallace and West[6] reported favorably on 12 abdominal fascial transplantations in nine cases of residual abdominal paralysis In cases of complete paralysis of the abdominal well anteriorly as well as laterally two operations were performed, in the first stage cross strips were run from the ninth rib to the opposite anterior superior spines on both sides, in the second stage rib cage and pelvis were connected by fascial strips laterally from the tenth rib to the crest of the ilium

3 The fusion

The definite procedure is the spinal fusion The best method and the one which gives the least percentage of pseudarthrosis is the method of Hibbs It consists in the formation of osteoperiosteal grafts which are placed to both sides of the spinous processes in the form of bone bridges and in the reaming out of the intervertebral articulations (Figs 43, 44)

How much of the spine should be fused? a) In paralytic scoliosis both primary and secondary curves should be included this means usually from the lowest horizontal vertebra above to the highest below the curve b) The sacrum should be included in all cases in which the lower border of the fifth lumbar is oblique One should be careful not to interfere with an adequate lumbar

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FIG. 44 (*top*) Paralytic scoliosis fusion. Case D B (female) #38-19724 13 years, June, 1938. Poliomyelitis at age of 10 years residual paralysis right lower and weakness of back muscles. Advanced progressive right lumbodorsal scoliosis Risser cast followed by two stage fusion from ninth dorsal to sacrum. *left* August, 1938, before fusion. *right* September 1947 eight years after fusion.

FIG. 45 (*bottom*) Paralytic lumbodorsal scoliosis fusion failed. Case D E. (female) *#38-15410* seven years, September 1932 Poliomyelitis six months ago marked paralysis left lumbodorsal scoliosis with oblique fifth lumbar 1936. Two stage fusion from eighth dorsal to fifth lumbar performed April, 1937 progressive in spite of fusion. Reasons 1) Dorsolumbar pseudarthrosis. 2) Fifth lumbar oblique but not fused to sacrum. *left* August, 1937 four months after fusion. *right* May 1942, five years after fusion.



FIG. 44 top FIG 45 bottom. (*legend on opposite page*)

II. PATHOLOGY

A. NEUROPATHOLOGY

The function of the different cortical motor areas and the subcortical ganglia has to some extent been established by animal experimentation When area No 4 is injured or ablated, the result is paralysis of the volitional movements but no spasticity There is a considerable increase of the deep reflexes but at the same time the extremity remains flaccid. If area 6, the premotor area, is removed the result is increased spasticity and the appearance of pathological forced grasping reflexes When superimposed upon a complete lesion of area 4, the destruction of area 6 is marked by rigidity in all muscles If the entire cortex is removed, the animal shows an increase in reflex tone of the muscles but it still maintains its erect attitude This increased tone maintains the fixation of the joints the attitudinal reflexes such as the neck reflex, and the general static reactions

The function of the lower subcortical centers which are supplied by fibers of the premotor area 6 and area 8 can be studied experimentally in animals by elimination of the ganglia. The neostriatum, that is, the nucleus caudatus and putamen are inhibitors of spontaneous movement, that is to say, they hold the movements in check by virtue of their connection with the cortical motor area The nucleus caudatus receives fibers from Brodman areas 4S and 8 and the putamen from Brodman area 4 The globus pallidus which receives fibers from area 6 is an inhibitor, and its stimulation will produce motion of a definite tonic or postural character It acts mainly upon the larger muscle masses and chiefly the axial muscles of the trunk and the proximal muscles of the extremities. Consequently elimination of the nucleus caudatus and putamen results in massive movement such as we see in athetosis and chorea. If the lesion involves the globus pallidus also, a so-called extrapyramidal rigidity develops a condition which is also represented in some cases of spastic paralysis.

Summary The flaccidity or muscle weakness indicates only a lesion of the central gyrus of the motor area. Spasticity indicates a lesion of the premotor area alone or in combination with the motor area of the central gyrus In lesions of the motor area alone there are increased reflexes such as the Babinski and Hoffman reflexes and the deep reflexes and there is loss of fine and rapid movement, or adiadochokinesis Lesions of the premotor area produce grasping reflexes Lesions and destruction of the nucleus caudatus and putamen cause athetosis. Finally lesion of the globus pallidus the paleostriatum, causes akinetic rigidity and loss of automatism

B MUSCLE PATHOLOGY

The normal muscle at rest receives no impulses from the central nervous system and therefore no action current potential can be recorded When it goes into voluntary contraction it shows a more or less even pattern of oscillation which is uniform in frequency and amplitude so long as the contraction

s maintained without change The normal muscle can also be trained to carry out moderately powerful innervation singly without concomitant innervation of a synergist or antagonist That is to say, a slow movement will allow changes of position without the so-called myotatic reflex

In the spastic muscle the situation is entirely different The muscle has an increased stretch reflex, therefore active as well as passive motion will produce definite electrical responses A reflex contraction in turn produces a stretch reflex in the opposing muscle This simultaneous activity of the antagonistic muscle is a characteristic and is the chief cause of the clumsiness and stiffness of the voluntary effort in the spastic There is a constant spreading of reflexes to muscles which are supplied by other spinal segments even on the opposite side The rigid muscle behaves similarly to the spastic, but the voluntary movements are slow and weak The action currents are the same as seen in the spastic except they are present, in lower intensity, also during apparent rest The result is a great tendency to synchronization and grouping of action potentials The tremor consists of rhythmic oscillations, five to 12 per second, separated by inactive intervals In athetosis again the management of the motor unit is similar to that we see in the normal muscle during voluntary con traction, but there is superimposed a synchronous, maximal activation of the synergistic and antagonistic muscles This produces a so-called tonic "fixation" Involuntary movements superimposed upon voluntary movements are the char acteristic feature of athetosis

III PATHOGENESIS

The three major groups are the congenital or prenatal type, the obstetrical type caused by birth trauma, and the acquired type from traumatic, circula tory or infectious causes occurring during life

A. THE PRENATAL TYPE

Little is known about the prenatal pathology, though autopsies have established a number of changes of the central nervous system in isolated cases of spastic paralysis such as the porencephaly, the internal hydrocephalus, certain congenital aplasias and amyelinizations of the white tracts Another condition is the so-called status marmoratus of the putamen, first described by Anton (1896), in which this structure appears as a polygonal lattice work of white fibers due to the influx of additional fibers from the frontopontine tract.

B THE BIRTH TRAUMA

The birth trauma as a cause of spastic paralysis has been greatly overrated It is true that a number of factors may produce intracranial hemorrhage, for instance premature or precipitated birth, protracted labor, excessive molding of the head by disproportions of the mother s pelvis or the application of for ceps On the whole, however, premature births and any other birth complica

tions are so much more frequent in comparison with spastic paralysis that these factors cannot be considered a common cause

C THE ACQUIRED SPASTIC PARALYSIS

The acquired spastic paralysis can be divided into four groups as follows 1) The *inflammatory post natal* group is represented by the infectious ensephalitis which may destroy the motor region of the brain. 2) The *circulatory* group covers hemorrhages into the internal capsule or other sites in the locomotor system, vascular degeneration and thrombosis with occlusion of vessels. 3) *Traumatism* may cause direct injuries to the brain or subdural hemorrhages which leave cysts or extensive scars 4) *Degenerative lesions* are the primary atrophy and shrinkage of the neostriatum and globus pallidus combined with that of the thalamus, this is observed, for instance, in Parkinson's disease, and in toxic degenerative necrotizing lesions of the basal ganglia such as follow carbon monoxide or barbiturate poisoning Others are due to avitaminosis or metabolic degeneration as, for instance, in Wilson's disease Degenerative lesions may also be caused by tumors and neoplasms such as gummas and tuberculomas

IV CLINICAL PATHOLOGY

A TYPES

1 The spastic type

This type is the most frequent. Of 1720 cases of spastic paralysis cases reported by Schwartzmann and McCarroll,[14] 1217 were of this group

2. The athetotic type

This type is characterized by involuntary and persistent movements, shifting irregularly from one muscle group to another The contractures are smooth, powerful and sustained The electromyographic studies of Putnam and Hoefer[15] show that in synergists and in antagonists there is almost constant, simultaneous innervation both during the voluntary and the involuntary movements. The abnormality is that the movements are not within the reach of volition, and there is none of the normal reciprocal relaxation of the antagonist. This extrapyramidal group constituted 225 of 1720 cases in the series of Schwartzmann and McCarroll.[14]

3 The ataxic group

The ataxic group cerebellar lesion is a small group as is also a fourth group that of *cerebrate rigidity*

B LOCATION

1 Spastic monoplegias

Only one limb is involved, usually one upper extremity These are the least difficult cases to manage because of the limited extent of the paralysis and also

because their intelligence is usually unimpaired (Fig 47) The characteristic trias of symptoms for the upper extremity consists in flexion deformity of the wrist, pronation of the forearm and adduction and inward rotation in the shoulder joint In spastic monoplegia of the lower extremity the attitude is characteristically the hip and knee in flexion and the foot in equinus position The knee is inward rotated and the adductors are contracted



Fig 47 Spastic monoplegia. Case L. L. (male) #38 14093 four years, September 1921 Congenital spastic monoplegia of right hand marked flexion and pronation contracture



Fig. 48. Spastic hemiplegia, right Case R. F (male) #44 5651 12 years, June 1944 Adduction shoulder flexion elbow flexion and pronation wrist. Adduction and inward rotation hip flexion knee equinovarus ankle.



Fig. 49 Spastic quadriplegia.

2. The spastic hemiplegias

In the spastic hemiplegias both the upper and the lower extremity of one side are involved but to different degrees The patient has more difficulty in locomotion than the spastic monoplegic with only the lower extremity involved, because he lacks the balancing swing of the upper extremity during the gait (Fig. 48)

3 The spastic quadriplegias

In the spastic quadriplegic we find that, aside from the disabilities of the upper extremity, the gait is severely handicapped The strongly adducted and inward rotated thighs render it most difficult to swing the limb forward past the standing leg In addition, the equinovalgus or varus contracture prevents any take-off of the feet, and the propulsion is an alternating swivel-like action around the ball of the standing leg (Fig. 49)

C SPASTICITY AND INTELLIGENCE

It is not easy to measure the intelligence of spastics by such psychometric tests as the Stanford Binet method. Single examinations are insufficient. It is necessary to measure the child s mental development repeatedly over a period of time We have found in our series of spastic cases that of 300 cases analyzed the intelligence was normal and above in only 26 6 per cent. The moron group comprised another 26 6 per cent, the imbecile group 23 per cent and the fourth, the idiotic group with the lowest intelligence about 24 per cent. In general, we find that the more extensive the spastic paralysis, the lower as a rule is the mental state. There are some exceptions Especially in the athetotic type one may find a high intelligence quotient even in severe cases In the monoplegic group we found normal intelligence or higher in 70 per cent of the cases In the diplegic group normal intelligence was found in 45 per cent in the para plegic and quadriplegic groups, however there was normal intelligence in only 7 per cent of the cases.

V TREATMENT OF SPASTIC PARALYSIS

Of the seven cases per 100,000 population only six survive the first year Phelps estimates that two of these are of such low intelligence that nothing can be done for them, while two of the milder cases can be left to themselves and do not need institutional treatment so that only two of the seven cases per each 100 000 need treatment. With the normal span of time for treatment estimated at 20 years it would mean that for each 100 000 population there are at least 40 cases who need medical care As the treatment is very largely one of training, the prognosis is closely associated with the intellectual status of the patient

It is quite obvious that the treatment of a condition as complex as spastic paralysis is not a matter of applying a single method. It requires a well laid plan comprehensive enough to meet many problems It must be realized that

all treatment is symptomatic. Since the avenues of normal motion are blocked by the permanent pathological changes, all treatment is also substitutionary, this means it consists in developing circuitous routes around permanently destroyed motor centers In the last analysis one must rely upon the versatility and docility of the motor centers to develop and utilize such bypaths to locomotion The background of treatment, therefore, is educational, all other methods, medicinal or operative, are merely incidental insofar as they prepare the way for training and re-education

A THE PLAN OF TREATMENT

The plan of treatment may be formulated as follows

1 The first postulate is alignment. It is obvious that the distorted limbs of the patient cannot be made to function until the normal relationship of the parts is restored

2 The second postulate is stability Whether this is provided actively by muscle balance or by braces or operatively, it is an essential prerequisite for any program of muscle training

3 The third postulate is relaxation This is largely though not entirely a problem of training It requires special provisions for rest and isolation, freedom from excitatory influences and involves an elaborate system of providing mental and physical repose

4 The fourth postulate is alternation It is essentially the treatment to overcome mass movement and to teach individual and well directed, properly timed motion The patient must learn to move the extremities or parts of them independently and free himself from concomitant movements. Alternation of the muscles by the use of bicycles is a point in this training In the athetotic it is much more difficult, because here activity is hampered by the superimposed voluntary motion

5 The fifth postulate is automatism i e teaching the patient to carry out motion without thinking It is a principle which rests on relaxation and alternation Whoever observes a spastic in action, as it were, will realize the enormously strenuous and fatiguing concentration with which he tries to carry out the simplest movement. The point is to lighten his burden, mental and somatic, by training him in an automatic performance of movements, which comes closer to the physiological conditions.

Of great help in acquiring automatism are the conditioned reflexes of the auditory and visual kind which take the place of the normal proprioceptive reflexes for instance, timing by voice, drum, metronome, phonograph, control of movement by floor marks or mirrors

Neurological operations have been used with indifferent success to produce relaxation Only alignment and stabilization can be achieved by orthopedic measures. All other requirements are strictly educational A certain appropriate program must be set up for the rehabilitation It is important to assign a special timing for all educational steps Phelps[13] recommends that under one year

one should try the gross motor coordination which involves the large joints. Then in the pre-school years from two to five, speech, standing, walking and behavior in general can be developed The correction of speech defects is a difficult problem One method is to have the patient imitate other children or let him enunciate and speak in front of a mirror where he can watch his face and tongue Specialistic training, however, for defective speech will be required *in all cases where the speech defect is* of any extent In the primary school ages, from five to ten, one should try to teach the final coordination of hands and feet, like cycling roller skating or skipping

B TECHNIQUES OF TREATMENT

1 Relaxation

a) RELAXATION can be achieved by *application of antispasmodics*

1) Hyoscine is a useful drug in postencephalitic conditions when the disease is contracted after the fundamentals of reciprocal innervation have been acquired (E W Phelps[11]) In the child who is injured at or before birth the administration of this drug is of no value

2) Curare This is extensively used and was first introduced by Burman.[1] It is now prepared in accurately assayed solutions for intramuscular and intravenous use Most common is the Intocostrin recommended by Kingbert[6] in doses of 1 cc. of a 10 per cent solution for every 40 pounds body weight, the maximum dose not to exceed 5 cc. intravenously The effect is very transitory and the dosage must be repeated

3) Quinine methochloride introduced by Bennett[1] and King[6] has a strong curare like action when given orally or parenterally in doses of 10 to 50 mg per kg body weight (Harvey[4]) It also wears off quickly The antidote is the same as for curare 1 cc. of 0 001 prostigmine

4) Dilantin sodium is also used as an anticonvulsant, and *it is a substitute* for phenobarbitol It is more useful in tension athetosis than in spasticity

5) Prostigmin. This drug destroys the cholinesterase, and we have already explained its physiological action in the treatment of poliomyelitis It is believed to be of help in the correction of contractures when used in connection with braces *or casts (Phelps[11]) All* antispasmodics have a temporary effect only

b) A MORE RELIABLE AND LASTING METHOD to secure relaxation is by physiotherapy, i e hot packs and warm pool baths There must be ample intermissions in the training program to avoid fatigue Massage has hardly any place in the treatment of spastics stretching of contracted muscles should be carried out very gently in order not to exaggerate the already increased stretch reflexes

c) RELAXATION BY NEUROLOGICAL OPERATION

Operations are merely an incident in the treatment plan of spastics, further more of all the features of spastic paralysis it is only the spasticity which is amenable to surgical treatment, if we except the operation on the central nervous system *itself for the control of the athetosis.*

1) The sympathetic ramisection was introduced by Royal and Hunter

(1923) under the concept that the sympathetic nervous system has some connection with spasticity and muscular rigidity It has been shown, however, that sympathectomy does not influence the rigidity and that it does not affect the plastic tone or the contractility of the muscle Its only function is the motor innervation of the blood vessels Sympathectomy is hardly ever applied today for the relief of spastic paralysis

2) In the operation of Foerster[5, 6] the posterior rhizotomy, the underlying idea is to cut the reflex arc and thereby destroy the abnormal reflex irritability seen in a certain type of case In our own cases we found it of questionable value

3) The cord operations for athetosis (Putnam[11]) The rationale is that abnormal movements are due to impulses which come down over the pyramidal pathways as the result of direct irritation (Kahler and Pick[7]), or according to the idea of Monakow,[12] they are the result of abnormal stimuli from the cerebellum or they come over the extrapyramidal pathways from the corpus striatum.[1]

Whatever the case may be, the operation consists in dissection of the extra pyramidal tracts above the brachial plexus One tract lies anterior to the crossed or lateral pyramidal tract in the lateral cord, and the other lies at the periphery of the cord in the anterior quadrant. It has been shown that destruction in this region does not produce any motor defect (Hindman) The tracts are cut at the level of the second or third cervical, and it is said that the operation is capable of providing substantial relief in mild as well as in severe cases of athetosis

4) The operation on the brain cortex or decerebration is an old operation first introduced by Horsley[8] who performed it for athetosis The extirpation involves the precentral regions 4 and 6 and is effective in abolishing the involuntary movements of the choreo-athetosis The operation has a comparatively low mortality and has lately been particularly advocated by Klemme,[10] who found 39 cases completely and 24 partially relieved and rehabilitated, among 100 cases reported.

Summing up these neurological operations we believe that the rhizotomy of Foerster is of value only in a small group of cases, those of markedly increased reflex irritability without athetosis The Putnam operation is of value in selected cases of athetosis and is a more reliable operation, although the question at this time is not decided It should be mentioned also that extirpation of the head of the caudate nucleus was carried out by R Meyer[11] through the anterior horn of the lateral ventricle with good results in several cases

2. Alignment

a) CONSERVATIVE

Traction is useful in overcoming contractures, especially of the knee joint. Usually the muscles yield readily to mechanical devices, particularly after they are relaxed by application of heat.

b) SPECIAL ORTHOPEDIC PROCEDURES FOR ALIGNMENT

1) The spastic equinus. It is necessary to ascertain whether the equinus appears only on weight-bearing as a reflex contracture, or whether there is in addition a structural shortening of the muscle In the first case the indication is the selective nerve resection as introduced by Stoffel [17] The popliteal nerve is separated into its different bundles, and the branches to the gastrocnemius and soleus are partially resected, removing as much of the motor nerve supply as one thinks necessary to restore the equilibrium However, if the tendo Achilles does not relax during sleep, then we know that there is a structural shortening in this case a lengthening of the tendo Achilles is necessary There is a great tendency for the shortening of the heel cord to recur in young chil dren because of the growth of the tibia It is for this reason that we prefer to wait with the lengthening of the heel cord until the child is four or five years old

2) The flexion contracture of the knee This is often associated with spastic equinus The correction of the deformity itself is mainly a matter of conservative treatment, particularly braces and casts The restoration of balance, however, often requires operative procedures If the hamstrings are spastic and the quadriceps weak, a good deal can be accomplished by tendon transplantation of the hamstring muscles after the contracture has been cor rected conservatively or by operative means If it is mainly a matter of a weak quadriceps, it may be sufficient to tighten the muscle by pulling the patella strongly downward and reimplanting the patellar tendon more distally into the tibia, a procedure first recommended by Chandler This is sufficient only for milder cases In most instances it will be safer to resort to the hamstring transplantation, using both hamstrings for implantation into the patella.

3) In the hip joint the most common contracture is flexion, internal rotation and adduction The internal rotation is caused mainly by the anterior half of the gluteus medius and minimus and the flexion mainly by the tensor fasciae In addition to this the adduction contracture is due to the hyperinnervation of the adductor muscles Here again the choice is between obturator nerve resec tion alone, or additional section of the adductors, according to whether the con tracture is due to hyperinnervation alone or to additional structural contracture of the muscles If the adductors relax completely during sleep and the spas ticity reappears on innervation treatment is resection of one or both branches of the obturator nerve They can be reached intra-abdominally by the opera tion of Selig or in bilateral cases by making the so-called transverse or Pfan nenstiel s incision or more simply by direct exposure of the branches at the thigh If there is both innervational contracture and structural muscle shorten ing i.e if the adductors do not completely relax during sleep, the obturator resection should be combined with section of the adductor muscles This is the more common situation *After resection of the obturator nerve and section of*

the muscles, the legs are put in strong abduction The cast should not remain more than four weeks, otherwise an abduction contracture may develop

Sometimes the internal rotation contracture of the hip which is associated with adduction persists after resection of the obturator nerve due to tension of the anterior fibers of the gluteus medius and minimus Durham recommends for this correction a simple operation for releasing the gluteus medius and minimus from their point of insertion at the greater trochanter However, we have observed in a number of cases that the operation weakens the abductory power considerably, and the Trendelenburg symptoms become strongly positive We believe that ultimately a subtrochanteric osteotomy with outward rotation of the femur would solve the problem without interfering with the abductory power of the hip The flexion contracture of the hip joint, so far as it cannot be fully controlled by stripping operations, can likewise be completely corrected by a subtrochanteric osteotomy

3 The spastic disalignments and imbalances of the upper extremity

The spastic shoulder The status of the external rotators determines the persistence of the deformity If they have any power left, some equilibrium can

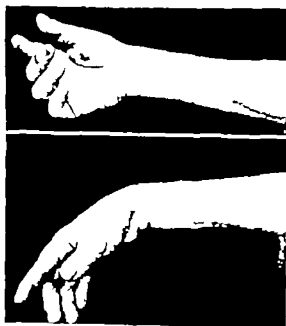


FIG. 50 Spastic pronation contracture flexor carpi ulnaris transplantation Case A. H (female) #38-30242 five years, July 1917 Spastic hemiplegia with pronation contracture of forearm. Transplantation of flexor carpi ulnaris to radius 1935 age 22 years active supination. Postoperative observation 10 years.

be established by tenotomy of the subscapularis and outward rotation of the arm. In case of paralysis of the outward rotators the better procedure is the osteotomy below the surgical neck followed by external rotation

Flexion contracture of the spastic elbow rarely requires operative measures

Spastic pronation contracture of the forearm is a difficult problem It is most disabling and requires surgical interference. First, the contracture must be overcome so that the forearm can be fully supinated passively Then active supination can be established by tendon transplantation For this purpose we have found our method of transplanting the flexor carpi ulnaris most satisfac tory The muscle is isolated full length from a long volar incision and then led obliquely over the dorsum of the forearm and inserted by drill hole into the lower end of the radius (Fig 50)

4 Stabilization

All joints of the lower extremity require a considerable degree of stability in the corrected position Braces have a definite place in stabilizing the joints of the lower extremity during the process of realignment or beyond, when the corrected position is to be secured Long leg braces have the added advantage in the lower extremity that their weight counteracts reflex spasm They give the patient the feeling of security and relieve him of the emotional strain of maintaining his joint in alignment.

For permanent stabilization, when active muscle balance cannot be restored arthrodesing operations are necessary The most common situations are as fol lows

Spastic equinus associated with varus or valgus deformity In all these cases in which the lateral balance is disturbed we find that the selective nerve resection is entirely inadequate The situation calls for a stabilizing operation which must include both the subastragalar and the midtarsal joints Such an operation, of course, should not be performed before the age of eight or ten, up to that time the foot must be held in control by braces Should a foot drop also be present because of weakened extensors of the foot, it is better to select *the Lambrinudi type of operation If there is marked clonus in addition to the spastic equinovarus, it will help to add the selective nerve resection to the* stabilizing operation

The spastic thumb The spastic so-called "underslung' thumb interferes with the grasping function of the hand As the fingers are being closed the strong action of the flexor pollicis longus pulls the thumb sharply into the palm Tendon transference (extensor indicis to extensor pollicis longus) gives only temporary results The only method we found reliable was to fuse both the carpometacarpal and the metacarpophalangeal joint of the thumb in proper position so that the thumb acts as a fixed post in precise opposition against *which the fingers can be moved* (Fig 51)

The flexion contracture of the wrist. It would seem a simple problem to transfer some of the strong spastic flexors to the extensor side and to secure the position by arthrodesis of the wrist. But the situation is not as simple as that. For one thing, the tendon transference of wrist flexors to finger extensors



FIG. 51 Spastic paralysis of hand spastic thumb Bone graft arthrodesis Case J R. (female) #40-6555 three years, October 1935 Pronation contracture right arm with "underslung" spastic thumb. Bone graft between first and second metacarpals, 1934 failed. Fusion metacarpophalangeal joint of thumb 1935 result fair Fusion carpometacarpal joint 1944 excellent result. Fingers can be closed without catching the thumb.

does not show the result we see in the drop wrist, because the innervational load of the muscle is in constant flux, and it is difficult to establish a stable equilibrium Furthermore, in cases in which the extensors are very weak or gone, the active flexion of the wrist is used for the opening of the fingers These kinetic conditions must be taken in consideration before an arthrodesis of the wrist is carried out (Fig 52) In other cases the extreme flexion of the wrist is not necessary to open the fingers, because there is enough strength in the extensors Here the arthrodesis of the wrist is indicated (Fig 53)



FIG 52 Spastic flexion contracture of wrist arthrodesis. Case L C (male) #C-8745 22 years, January 1929 Spastic hemiplegia left strong flexion contracture left wrist straightened passively Arthrodesis March, 1929 good position of wrist 1932 but patient unable to extend fingers or release objects.

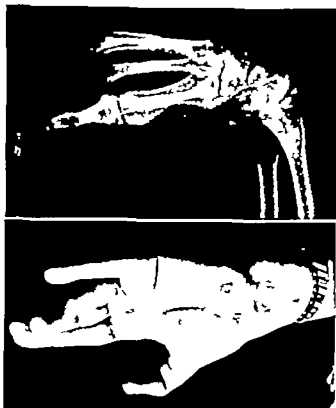


FIG. 53 Spastic flexion contracture of wrist transplantation and arthrodesis. Case D D (male) #38-10611 18 years, August, 1937 Spastic hemiplegia marked flexion contracture of left wrist. Transplantation of flexors of wrist to extensors of fingers arthrodesis of wrist. Good position good flexion and extension movement. Observation 11 years. a) Before operation. b) 1948, 11 years after operation.

C REVIEW OF INDICATIONS AND RESULTS OF OPERATIONS FOR SPASTIC PARALYSIS

1 Anatomic results

a) Instability of foot and ankle are best taken care of by arthrodesing operations Good results were obtained in 62 5 per cent.

b) Flexion contracture of the hip corrected by the Soutter operation gave good results only in 58 per cent, and in many cases it was necessary to add the subtrochanteric osteotomy

c) Tendon transplantation on the whole gave poor results, with the exception of the transplantation of the hamstrings to the patella for flexory imbalance of the knee and transplantation of the flexor carpi ulnaris dorsally to the radius to provide active supination. The latter operation gave good results in 66 per cent.

d) The adductor tenotomies with or without obturator resection gave good anatomic results in 63 per cent of the cases

2. Functional results

Only about 22 per cent of all spastic cases were operated. In general one finds that the alignment is comparatively easy but that it is more difficult to provide active motion So far as it is possible to obtain active muscle balance long periods of muscle training and re-education are required For the weight bearing joints especially foot and ankle stabilizing operations are necessary

The mental status of the patient is an important factor for the success of a prolonged training program, not only in the conservatively treated cases, but also in postoperative care This is an added reason why the functional results are far better in the monoplegic and diplegic types (50 per cent) than in the paraplegic type (25 per cent)

Taking the spastic group as a whole and considering all operations and all situations as well as all types of spasticity and all degrees of mentality, we find the total of good functional results to be 36 per cent, to which 28 8 per cent could be added as showing considerable improvement This gives a total of 65 per cent of all operated cases in which the end result justified the intervention

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SECTION B

STATIC DISABILITIES

Lecture I

ON LUMBOSACRALGIA OR LOW BACK PAIN

I ORIENTATION

LOW back pain is a highly controversial subject The argument centers not so much about observational facts as about their interpretation in pathological terms It only adds to the difficulty that low back pain is so often a symptom of remote disorders which are not directly related to the structures composing the lumbosacral region The diagnostic analysis requires first to segregate the low back pain which is a symptom of a remote cause, from the so-called idiopathic type and then to interpret idiopathic backache on the basis of established biological facts

A THE SYMPTOMATIC LOW BACK PAIN

1 General infections

General infections such as typhoid, acute exanthema, influenza, smallpox, acute articular rheumatism, or septicemia are not uncommonly associated with pain in the back Here we rely on general systemic symptoms accompanying back pain to establish the diagnosis

2. Localized lesions

In localized lesions of other systems which produce low back pain, it is more difficult to recognize the symptomatic character of the pain For instance, appendicitis infected and enlarged retroperitoneal glands, hemorrhoids and diseases of the colon and rectum may produce pain simulating idiopathic lumbosacralgia. Affection of the genito-urinary system such as pyelitis, stones or tumors renal disease, diseases of the ovaries or uterus, particularly mal position and adhesions, and tumors of the prostatic gland are frequently associated with low back pain In these conditions the back pain is not dependent upon motion and posture of the body as is the idiopathic low back pain This fact, together with other manifestations of dysfunction facilitates the diagnosis Diseases of the central nervous system produce backache, for instance, meningitis lateral sclerosis or tumors of the cord (see intervertebral disc) Intense pain in the lower back may also be caused by tumors of the vertebral column especially metastatic malignancies, and by myeloma

B IDIOPATHIC LOW BACK PAIN

The idiopathic low back pain in the stricter sense is a definite clinical entity It is produced by injuries to the soft tissues of the lower portion of the spine, a mechanical derangement of these tissues

II. PATHOGENESIS

It is essential to establish the following points a) The identity of the soft structures involved in mechanical disorder and how each responds to sprains or strains. b) The anatomical anomalies which cause these structures to become more susceptible to mechanical strain c) Pathological conditions of the spine which facilitate strain d) The characteristic pain patterns which these structures develop under strain e) The effect of strain of these tissues on the neighboring spinal nerves

A THE STRUCTURES UNDER STRAIN

The structures involved are the short and long ligaments of the spine, all capsular reinforcements, all deep and superficial long and short muscles, and the nervous structure within and outside the spinal canal

In pathological terms a ligamentous strain is a rent or tear followed by fibrous repair Repeated strains cause a permanent loss of the elasticity and resiliency of these structures A muscle strain is a tear within the muscle or at its attachment It is also repaired by scar tissue. The muscular tissues of the lower back respond to strain primarily by developing spastic contractures which may impose upon the body a certain pathological attitude or posture. Stress or pressure upon a peripheral nerve causes fibrosis of the endoneurium and perineurium and axis cylinder degeneration, its immediate response to mechanical pressure is of the neuralgic type and is followed by motor deficiencies such as decrease of reflexes

Excessive flexion movements cause sprain of the soft structures in the following sequence (Haboush[17]) The iliolumbar ligament is strained first. Then the check is taken up successively by the fourth, the first, second, and third lumbar interspaces. At the limit of flexion the supraspinous ligaments and the lumbar fascia act as a check. In rotary movement and in side bending the sequence of strain is first, to the iliolumbar ligaments, then to the quadratus lumborum and the intertransverse ligaments on the convex side Excessive side movement strains or tears the quadratus lumborum, then the strain extends to the lateral fibers of the longitudinal ligaments the ligamentum flavum and interspinosum and finally it separates the neural arches. A healthy disc usually remains intact in forward flexion and in side bending

B CONGENITAL VARIATIONS AND ANOMALIES PREDISPOSING TO LOW BACK STRAIN

1 Variations in the anatomic build of the spine

The slender anatomic build is especially prone to develop malposture because of the generally decreased muscle tone. The flat lumbar spine often associated with this type is unable to resist static stresses transmitted from above The hollow back with its accentuated lumbar lordosis horizontal

sacrum and a wedge shaped fifth lumbar vertebra is under an abnormal forward shearing stress at the lumbosacral junction which easily results in strain (Von Lackum)

2. Congenital anomalies affecting mobility

The arrangement of the lumbosacral articulation may vary from the normal, being oriented in the sagittal instead of the frontal plane on one or both sides (*Tropism*) This deprives the junction of its normal rotatory range, and it cannot absorb the rotatory impulses coming from the upper body or through the pelvis. An *abnormally long transverse process* or, in its higher degrees, the *sacralization* is another variation which reduces mobility at the sacro lumbar junction (Fig 54) In this case the motion in the sacrolumbar region becomes transferred to the articulation between the fourth and fifth lumbar, since this articulation is usually arranged in the sagittal plane, lateral motion and especially rotation suffer a considerable restriction At one time this anomaly was believed to be responsible for sciatic radiation (Bertolotti and Schiassi) Sacralization is a frequent anomaly, being found in 3 8 per cent of all spines, more often bilateral than unilateral, yet in the majority of cases it does not produce radiation One cannot therefore interpret sciatic radiation on the basis of sacralization even though the anomaly may well be a factor predisposing to mechanical strain



Fig. 54 Sacralization.

3 Congenital anomalies affecting stability

To this group belong the different *clefts* and *pseudarthroses* the incomplete formation of the laminae in the lumbar and sacral regions, the so-called *separate neural arch* and the forward displacement of the fifth lumbar vertebra upon the sacrum, the *spondylolisthesis* These anomalies are not directly responsible for the low back pain, as is shown by the fact that one sees many cases of spondylolisthesis without either low back pain or radiation, nevertheless, such a spine is potentially weak and susceptible to strain because the ligamentous apparatus at the junction is poorly developed Low back pain has also been ascribed to the opposite deformity or *retrospondylolisthesis*, the backward gliding of the fifth lumbar, the occurrence of which is denied by Willis We have not been able to find a case in our clinic (H Swerdloff) in which this condition could be proven when accurate x ray measurements were applied (Garland and Thomas[11]) Narrow intervertebral discs and Schmorl's

nodes can be seen in x rays of normal people as often as in cases of low back pain (*Brav, Bruck and Fruchter*[3])

C PATHOLOGICAL CONDITIONS OF THE SPINE WHICH PREDISPOSE TO SACROLUMBAR STRAIN

The most common condition is the *arthritic spine* The loss of motion at *higher levels is apt to throw an extraordinary strain upon the sacrolumbar* junction. This is particularly true in the Marie-Strümpell form of atrophic arthritis. *Spinal osteoporosis* likewise predisposes to strain, not only because of the accompanying osteoarthritic changes but also because of the general relaxation and lack of tone of the ligamentous and muscular structures *Deformities resulting from fractures at the dorsolumbar level lead to lumbosacral* lordosis which places excessive stress on the lumbosacral junction

D CHARACTERISTIC PAIN PATTERNS OF THE DIFFERENT STRUCTURES

1 Local pain

One must expect sensory responses to vary according to the sensory supply of the tissues The posterior primary division of the spinal nerves supplies the

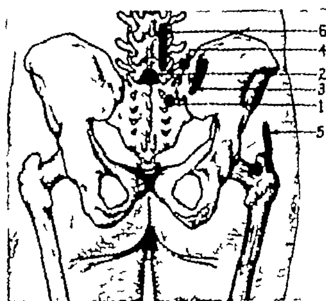


FIG. 55 (see text)

long and short muscles of the back their insertions, sheaths and aponeuroses, as well as ligamentous structures of the back. The highly sensitive ligamentous structures and the aponeuroses give a more acute response and produce localized pressure or "*trigger*" points. The muscles have a poorer sensory supply and they respond by diffuse pain manifestations and by reflex contracture

Stereotype pain patterns are the following (Fig. 55)

a) The Sacrospinalis Insertion Syndrome (Fig 55, 1)

The pressure point is at or near the posterior and superior iliac spine It indicates strain of the sacrospinalis insertion.

b) The Lumbosacral Syndrome (Fig 55, 2)

It exhibits a midline pressure point at the lumbosacral junction It indicates strain or stress of any of the ligaments at the junction of the lumbar spine with the sacrum

c) The Gluteal Syndrome (Fig 55, 3)

It has its trigger point at the origin of the gluteus maximus and indicates strain, tear or tension of the gluteus maximus aponeurosis

d) The Transversosacral Syndrome (Fig 55, 4)

It is caused by impingement of a large transverse process on the sacrum It has its trigger point near the tip of the fifth lumbar transverse process

e) The Tensor Fasciae Syndrome (Fig 55, 5)

It is due to strain of this muscle and causes tenderness along the fascia lata. It is often associated with abduction contracture of the tensor fasciae (Ober sign)

f) The Myofascial Syndrome (Fig 55, 6)

It denotes strain of the sacrospinalis muscle. There is no discrete trigger point but only diffuse tenderness along the muscle

2 The most acute response is that of a sensory nerve directly injured by pressure or pull This is called referred pain. A common example is the referred pain from intraspinal tumors or from a herniated intervertebral disc.

It is produced by direct pressure on the peripheral nerve at any point along its pathway If the pressure is proximal to the point where the spinal nerve bifurcates into its posterior and anterior primary divisions, the radiation covers both the back and front parts of the dermatome This bifurcation occurs immediately after the nerve has left the intervertebral foramen, except in the sacrum where there are separate foramina for the posterior and anterior primary divisions The radiation corresponds strictly to the anatomical distribution of the peripheral nerve, and it consists of both protophathic and epicritic sensations, such as paresthesias, anesthesias and burning sensations Inside the spinal canal pressure is most often caused by a tumor or a herniated disc, within the intervertebral foramen, it is usually due to an acute inflammatory condition or to arthritis

3 The third type of pain is the reflex pain Here a peripheral stimulus is conducted to the spinal cord, it becomes synaptic with another sensory center which in turn projects the pain peripherally along its sensory pathway (Fig 56)

We recall that this type of pain is produced by peripheral irritation, tha reaches the spinal cord by way of the somatic sensory fibers, and that it h activates a second sensory territory by way of synapsis Even though so orthopedic surgeons refuse to this day to accept the reflex mechanism a cause of sciatic radiation, the idea that radiating pain may be of reflex nat

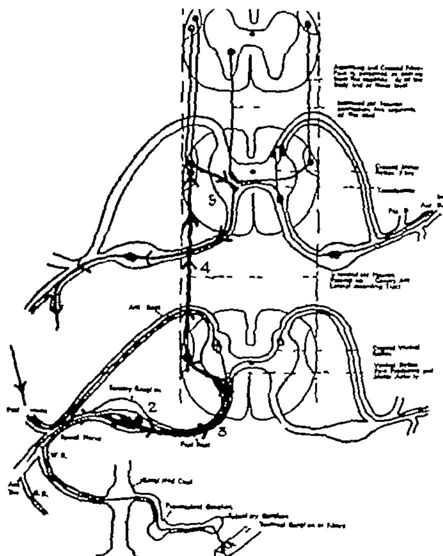


FIG 56. The pathway of reflex pain. Peripheral sensory stimulus (1) reaching spinal ganglion (2 and posterior root (3) becoming synaptic with intermediate neuron (4) ascending in Gower anterolateral tract and becoming synaptic with second sensory unit (5) and projected throug posterior root and spinal ganglion to periphery

is by no means new There is definite experimental evidence that the loca irritation of the structures of the back can actually cause radiations of purely neuralgic character in territories which have no anatomical connection wit the original pain producing stimulus Kellgren[19] found that if the interspinous ligaments are irritated between the fifth cervical and the third thoracic spine pain radiation appears in the dermatomes from the fifth cervical to the first

dorsal in *front and back* Irritation of the dorsal interspinous ligaments also causes *front and back* radiation in the dermatomes from the third dorsal to the fifth lumbar Irritation of the lumbar interspinous ligaments causes the same radiation from the third lumbar to the second sacral dermatomes In clinical medicine we meet with many instances of reflex radiation There is the reflex radiation to the left arm, shoulder and chest in coronary lesions, or the reflex pain between the shoulder joints in gall bladder disease, a similar reflex pain is observed in some cases of sciatic radiation associated with low back strain

III THE CLINICAL SIGNS OF LUMBOSACRALGIA

A PAIN

Local pain can be elicited either directly by pressure upon the trigger points as described above or, indirectly by 1 *Forcible compression* of the iliac crest if the origin of pain is the sacroiliac region 2 The so-called *Patrick sign* (Fig 57) Pain is elicited by pressure of the femoral head against the anterior portion of the capsule as the thigh is placed in flexion, abduction and outward rotation, and the knee is pressed against the table, the strain thereby being transmitted to the sacro-iliac junction 3 The *Lasegue or straight leg raising sign* Pain is elicited in the sacro-lumbar junction, and sciatic radiation is increased by backward tilt of the pelvis (Fig 58) The *Genslen sign* When the unaffected leg is strongly flexed and pressed against the chest, and the hip joint on the affected side is hyperextended, pain is caused by strain of the affected sacro-iliac junction (Fig 59)

B RESTRICTION OF MOTION

The cause of the restriction is the spasm of the muscles which automatically protect the injured region The spasm forces the patient to assume a position of relief, either a forward flexion or an inclination to the non affected side, the sciatic scoliosis (Fig. 60) He bends forward or backward by moving the spine as a whole all movements are carried out in the hip joints In side bending the opposite side of the pelvis is pulled up and the limb is lifted off the ground

C RADIATION

Sciatic radiation will be discussed more fully in connection with the herniated disc Not all radiation is due to herniation of the disc, however, nor does all radiation follow the territory of the sciatic nerve Lumbar radiation into the groin and the outer side of the thigh is caused by pathological changes in the upper lumbar and lower dorsal spine

D X RAY EVALUATION

For the interpretation of low back pain the x ray is of definite value only insofar as it reveals predisposing factors such as congenital abnormalities,









FIG 60. Sciatic "scoliosis."



FIG. 57 (*top*) Patrick's sign.

FIG. 58 (*middle*) Lasegue's sign.

FIG. 59 (*bottom*) Gaenslen's sign.

concomitant arthritis or traumatic changes The narrowing of the intervertebral foramen may be significant. It has been pointed out that the nerve roots are only slightly smaller than the foramina themselves so that even a moderate swelling of the capsular ligaments or ligamenta flava may cause compression of the roots in the foramina (Larmon " Magnuson") In addition, degenerative changes which flatten the disc, are liable to narrow the lumen of the intervertebral foramen. In fact a number of pathological changes may inter

fere with the free passage of the spinal nerve after it has left the spinal canal, and the view that all radiating pain is due to lesions of the intervertebral discs can hardly be supported (Key[20])

IV THE INTERVERTEBRAL DISC

A THE ANATOMY AND PHYSIOLOGY

That a portion of the intervertebral disc occasionally herniates into the substance of the neighboring vertebra or into the spinal canal is an old observation Kocher is credited with having mentioned it first in 1906 (Lewin[21]) The problem is so intricate that a review of the anatomical and physiological properties of this structure seems essential The disc is an elastic plate which consists of a peripheral fibrocartilaginous annulus fibrosus and of the semisolid nucleus pulposus in the center It is intimately connected with the bodies of the vertebrae by layers of hyaline cartilage, and it is closely interwoven with the anterior and, to some extent, the posterior longitudinal ligament Acting as an elastic cushion against perpendicular stresses, it is at the same time a universal joint in which all movement between the bodies of the vertebrae takes place It is practically avascular and nerve endings are extremely scarce in the disc itself, except in the posterior portion of the annulus They are abundant in the posterior longitudinal ligament (P G Roofe[22]) It has been reported that the patients in whom the disc was removed under local anaesthesia complained of severe pain while the annulus was being dissected (Spurling and Grantham[23]), but this did not prove that the pain originated in the disc itself

B THE PATHOLOGY OF THE DISC

1 Congenital malformation

A *persistent corda dorsalis* and portions of hyaline cartilage may be found in the vertebral bodies Congenital distortion of the disc occurs in *spondylolisthesis*

2. Traumatic changes

These changes are rare because the resistant cartilage plates secure the continuity of the disc. The posterior portion of the annulus bulges under stress and strain but, as long as the disc is intact, this bulging is only momentary

3 Physiological degeneration

The physiological degeneration of the disc begins very early and the desiccation of the nucleus increases rapidly with age In the second decade the nucleus begins to break up, it becomes more irregular while the annulus fibrosus still shows tightly packed lamellae. In the third decade, when the hyaline cartilage has already ceased its growth function, the annulus shows definite signs of wear and tear These retrogressive changes increase so that in

the fifth decade there are numerous protrusions of the nuclear disc substance through defects in the cartilage plate Finally the disc shows complete loss of structure, and most of its mass is now composed of fibrocartilage containing calcium deposits (Coventry, Ghormley and Kernohan,[8] Dencher and Love[10]) Deposition of calcium both in the annulus and nucleus, which accompanies these degenerative changes, was observed by Luschka, in 1858 Marrow tongues from the cancellous vertebral bone wander through the cartilage clefts into the disc and produce a complete fibrotization. In exceptional cases even a bony replacement of the disc is observed

4 Protrusions of the disc in general

Due to its nuclear turgor the disc tries to expand and it may hollow out the atrophic vertebral body, as in the osteoporotic spine. The vertebra assumes a characteristic fish tail shape In contrast, a pathological thinning of the disc is partly due to increased desiccation, as in old age, or it is due to actual penetration of the disc substance into the cancellous bone of the vertebra in form of Schmorl's nodes.

5 The posterior herniations of the disc

The first published report of a patient operated for protrusion of the inter vertebral disc into the spinal canal seems to be that of Oppenheim and Krause, in 1909 Cases were reported in the United States by Goldthwait,[11] in 1911 Calvé's and Galland s[4] report on the posterior protrusion of the disc was preceded by one of Mixter and Barr, who in 1922 called attention to unilateral leg pain as the outstanding symptom of a protruded disc.

It is interesting to note that, according to Beadle, not less than 15 2 per cent of all spinal columns examined showed such protrusions, and in 96 per cent they were located in the lumbar region (Love and Walsh[18]) In all spines examined by Schmorl, 15 per cent showed posterior herniations, all were in persons over 30 mostly in people over 50 years of age Associated with these protrusions is always a loss of substance with a thinning of the disc, although *some nuclear material remains* As a rule, the nodes do not attain sufficient size to compress the cord.

What, then are the special conditions under which such a posterior protrusion produces pain? According to W Duncan and T I Hoen,[11] the herniated discs may be devided into three types *The simple or fluid type* herniation appears at operation as a tense and round bulge of tennis ball consistency In the second type the capsule is ruptured, and part of the disc has entered into the extradural space In the third type the protruded portion of the disc is adherent to the nerve root In addition the herniated mass shows *considerable* edematous swelling owing to its ability to absorb water In older persons in whom the disc is degenerated and has assumed a fibrotic character, the capacity of the nucleus to swell is markedly decreased The clinical picture is obviously different in the three types and it is possible from the history to predict the

type of herniation to be found at operation A so-called concealed disc (Dandy[*]) is assumed in cases of intermittent sciatic radiation On certain movements of the spine the concealed disc retracts and radiation symptoms disappear This is possibly the explanation for the forward bent position assumed by the patient The completely herniated disc which is already adherent to a nerve root is not likely to allow these intermissions

C THE X RAY INTERPRETATION

1 The narrowing of the lumbosacral space does not necessarily imply a pathological process, because it may exist as a congenital condition without any clinical significance (Vinke and White[15]) Bray, Molter and Newcomb,[3] examining over 500 roentgenograms, found narrowing of the fifth lumbar disc at the posterior margin in 26 4 per cent and of the fourth lumbar disc in only 3 6 per cent, but the incidence of back and leg pain in these cases was not significantly greater than the remaining percentage not showing a narrowed disc. The conclusion is that the narrow fifth lumbar disc cannot in itself be considered clinical evidence of posterior disc protrusion



Fig. 61 Bilateral protrusion in the myelogram.

2 The Herniated Disc in the Myelogram (Fig 61) The ideal medium for visualization of the disc is pantopaque or iodized oil of low viscosity The myelogram is of value not so much for the diagnosis as for the localization of the disc and particularly for the recognition of multiple discs Spurling and Grantham[14] use it only in those patients in whom a strong suspicion of herniation exists without clear-cut neurological findings

D THE CLINICAL SYMPTOMS OF THE POSTERIOR HERNIATION OF THE INTERVERTEBRAL DISC

1 History

The usual history is one of long continued backache with recurrent attacks of radiating pain. One should ascertain, first, whether a trauma such as a fall on the buttocks, or a jar or a lifting strain has preceded the attack, secondly, whether coughing and sneezing aggravates the radiation as well as the back pain and finally, whether the radiation follows the sciatic root to calf and ankle and is accompanied by cramp-like feeling and paresthesias, as tingling and numbness The maximum pain intensity is in the gluteal region, the posterior thigh and leg. The pain is usually unilateral, although it may occasionally shift to the other side

2 Physical examination

The physical examination consists of a number of tests to establish sensory and motor disturbances

a) Sensory

1) Local tenderness may be found on percussion of the spinous processes of the third, fourth or fifth lumbar vertebra

2) Aggravation of pain in the affected leg due to increased intraspinal pressure is ascertained by the jugular compression or *Naffziger's test* According to Spurling, *a positive jugular compression pain is pathognomonic of an intraspinal lesion*

3) Anesthetic or paresthetic areas in the territory of radiation are recognized by pin prick and cotton touch examination Sensory loss on the top of the great toe indicates herniation of the fourth lumbar disc with involvement of the fifth lumbar root, loss at the lateral aspect of the foot particularly the lateral two or three toes, suggests herniation of the fifth lumbar disc with involvement of the first sacral root. Finally, sensory loss on the medial aspect of the foot appears in involvement of the fourth lumbar root.

b) Motor

Signs of motor restriction are, in addition to those mentioned in low back disorder Raising of head causes flexion in hips and knees (*Brudzinski test*) Flexion of knees with patient in prone position causes raising of the buttocks (*Ely test*)

c) The reflexes

A lesion in the third interspace involving the fourth lumbar nerve root causes abolishment or diminishing of the *knee jerk* A lesion at the fourth interspace involving the fifth lumbar nerve causes reflex change of the *posterior tibial tendon* Absence of the *Achilles jerk* is found in lesions of the lumbosacral disc.

Summary *A herniated disc in the third lumbar interspace* produces tenderness and radiating pain on percussion lateral to the third spinous process, a positive jugular compression test with tingling in the great toe a diminished knee jerk, and normal or only occasionally diminished ankle jerk, an absent posterior tibial reflex and hypesthesia or paresthesia in the fourth lumbar dermatome. A herniated disc in the *fourth lumbar interspace* with involvement of the fifth lumbar root causes sensory loss of the great toe absent or diminished knee jerk and pressure tenderness lateral to the spine of the fourth lumbar vertebra. A herniated disc in the *fifth lumbar interspace* involving the first sacral nerve produces tenderness to percussion lateral to the fifth lumbar spinous process, radiating percussion pain a positive jugular compression test with tingling on top of the foot and the lateral three toes, a diminished or absent ankle jerk, and hypesthesia or paresthesia in the first sacral dermatome.

3 Statistics on the frequency of clinical symptoms

According to Love and Walsh,[3] discs occur with greatest frequency in the lumbar region where they are found in 90 per cent of the cases, 98 per cent of the lumbar discs occur in the fourth and fifth lumbar interspaces Multiple discs are found in 12 per cent of the cases (G A Love[17]) Fifty-eight per cent of the cases with established discs give a history of specific injury to the back Sciatic radiation is found unilaterally in 78 per cent of the cases and bilaterally in 16 per cent Subjective paresthesias are found in 50 per cent Coughing and sneezing pain is observed in 55 per cent (Smith, Dery and Hagman[11]), and sciatic scoliosis in 39 per cent, objective loss of sensation is observed in only 21 per cent (Love and Walsh[23])

E LABORATORY FINDINGS

The laboratory findings add very little to the diagnosis Blockage of the canal is indicated by the rise of intraspinal pressure (*Quackenstedt test*) and by the increased globulin content in the spinal fluid None of these signs is even approximately reliable, and blockage is not necessarily associated with herniated disc symptoms

V DIFFERENTIAL DIAGNOSIS

A REFERRED VERSUS REFLEX SCIATIC PAIN

In cases of low back pain and sciatic radiation it is possible with the aid of the *novocain test* to distinguish between the referred and reflex types of radiation, provided a "trigger" point is present. The reflex nature of the radiation should be suspected when the pain is purely neuralgic, that *is*, *if* it carries no other sensory qualities except protopathic pain The novocain test is positive when injection of procaine into the "trigger" point area is followed by immediate and complete suppression of the reflex radiation and the disappearance of leg signs. To be considered positive the effect of this test should be striking and complete, no ambiguity should be accepted The patient must volunteer the information that both the local and the radiating pain have completely disappeared Probably not more than 10 per cent of all cases of sciatic radiation are due to reflex pain It is important, nevertheless, to recognize that reflex sciatica exists, and in doubtful cases the novocain test should be part of the clinical examination

B INTRASPINAL CONDITIONS OTHER THAN HERNIATED DISC CAUSING REFERRED PAIN

A neoplasm of the cauda equina may produce pain radiation very similar to that caused by a protruded disc The outstanding initial symptom *is* pain over the lumbosacral region radiating from the low back down one or both legs and consistently accompanied by weakness of the leg muscles The lipiodol

myelogram and the spinal fluid findings decide the diagnosis The latter are particularly helpful, because the total protein is often increased very substantially and there is xanthochromia

Other intraspinal lesions which cause compression in the spinal canal are extradural inflammatory lesions such as tuberculosis, tuberculomas or pyogenic abscesses from osteomyelitis of the spine Of the metastatic lesions, carcinoma of the breast and prostate head the list. Intramedullary tumors which cause *radiating pain are mainly primary tumors of the cord such as neurofibroma or* endothelioma but there is always the possibility of a metastatic lesion within the cord itself Of the primary tumors of the cauda equina and the filum terminale by far the most common is the so-called ependymoma.

The pain caused by intramedullary lesions of the cord usually can be distinguished from that due to irritation of the posterior nerve roots and ganglia It is a so-called central pain which is produced by irritation of the spinothalamic pathways *and is of burning or boring character There is a marked loss of* different sensations below the level of the lesion, together with hyperactivity of the tendon reflexes In intraspinal lesions which compress the nerve roots and cause radicular irritation the pain is usually sharp and is aggravated by coughing and sneezing or by flexing the head sharply against the chest (Craig and Walsh[7])

C THE EXTRASPINAL OR INTERVERTEBRAL TRUNCULAR PAIN

Most of these cases are *arthritics* The x ray picture may demonstrate osteophytes protruding into the intervertebral foramen Other signs are the narrowed



Fig 62. Osteoarthritis of a vertebra with sclerosis and irregularity of the facets.

intervertebral disc and the irregularity and sclerosis of the articular facets (Fig 62) In *spondylolisthesis* a certain percentage of cases will show sciatic radiation, but the number is small In our series of over 300 cases, it was found in only 4 per cent (Puig Guri[18]) The reason is possibly that the herniation, if it does occur, is in the midline and does not engage a sensory root

VI THE TREATMENT OF LOW BACK PAIN

A CONSERVATIVE

1 Immobilization and rest

In the localized low back strain due to injury of the soft structures immobilization can be expected to control the pain and to lead to the repair of the injured structures The routine program is bed rest, strapping or traction, and application of heat during the acute stages of the attacks. When the patient leaves the bed, his back is supported by a brace (Fig 63) Crutches are used in the beginning of ambulation Repeated attacks cause the ligamentous structures to become so relaxed that external immobilization is no longer able to restore their natural elasticity In this event the immobilization by external appliances gives only temporary relief operative measures must be resorted to



Fig. 63 Low back brace.

2. Mobilization

Not all symptoms of low back pain are due to injury of the ligaments, aponeuroses or fascial structures Some of them are caused by *reflex spasm* The control of these spasms requires stretching and manipulation This is well demonstrated in the cervical arthritis where the spastic neck muscles respond promptly to traction There is, therefore, in the treatment of low back pain a proper place and a proper time for either immobilization or manipulation, according to the structure which presents the dominant symptoms, as a rule, manipulation must wait until the recovery of ligamentous structures has been achieved by immobilizaion

Muscle spasm will often disappear spontaneously when the primary ligamentous lesion has healed We recall in this connection the maxim of Putti, who treated his cases of low back strain and sciatic scoliosis by immobilization

alone He put the patient in plaster in the pathological position, expecting that with the disappearance of the ligamentous strain the muscle spasm would also subside spontaneously and the deformity would disappear The particular technique of manipulation seems to be of lesser consequence than the proper time of application A number of methods are advised (Lewin[a]) They all consist essentially in the stretching of the back muscles either by flexion of the thigh on the abdomen or by carrying out flexion, extension and rotation of the body with the patient prone or on his side.

B OPERATIVE

1 Internal fixation in cases of sacrolumbar relaxation

In cases of persistent backache which do not respond to any method of external immobilization, one is justified in accomplishing internal fixation by a fusion operation This applies particularly to the habitually unstable spine with permanent relaxation of ligaments and a thinned out fifth lumbar disc.

2. Specific operations for conditions other than lumbosacral relaxation

a) Impingement of Spinous Processes

Impingement of spinous processes calls for their resection In these cases the supraspinous ligament is sensitive to pressure, and strictly localized pain is elicited on backward bending The origin of pain can be determined accurately by a preoperative procaine injection.

Case A.M—Male Adm December 1931 Age 34 yrs.
Pain at sacrolumbar junction after fall, for 11 years Tenderness between fourth and fifth lumbar spines. Temporary relief by procaine. Complete and permanent relief after resection of the impinging spinous processes of fourth and fifth lumbar

b) Transversosacral Arthritis

The strain arises from the irritation of the capsular ligaments of the articulation between the enlarged transverse process and the sacrum. In these cases resection of the *transverse process* is justified

Case W.A.—Male *Adm* August, 1935 Age 18 yrs.
Insidious onset. Tenderness left transversosacral joint. Radiation posterior left thigh and leg. Positive leg signs on the left. Marked shift to the right. Novocain injection at the left sacralized transverse process gave relief for one hour with disappearance of leg sign. Patient was completely relieved by transversectomy of *the left transverse process of the fifth lumbar*

c) Persistent muscular spasm

Many years ago Freiberg maintained that sciatic radiation was due to contracture of the piriformis muscle, since the patient was unable to rotate the limb inward. He advised the myotomy of the piriformis Ober [30] on the other hand, contended that the cause of the radiating pain lay in contracture of the tensor fasciae and therefore advocated the section of the fascia lata Heyman [a]

released the sacrospinalis spasm by sectioning the long muscles of the back These are all reflex contractures secondary to lesions of ligamentous or tendinous structures The operations are justified in cases in which the spasticity persists after the ligamentous strain has disappeared and in which it does not yield to conservative measures such as stretching or physiotherapy

The patients are strikingly relieved by the Heyman or the Ober operation, provided the indication is made on the basis of a persisting spasticity The contraction of the tensor fasciae, the so-called *Ober sign*, should be considered as pathological if there is also stress tenderness along the fascia lata, and in this case only is the operation justified

In his report on 86 cases, Ober[*] cites 41 excellent results and 33 cases of improvement. The indication for Heyman's[1] operation is similar, only in this case the contracted muscle is the sacrospinalis He reports 77 per cent of the cases with complete relief In our series of carefully selected cases in which the Ober operation was performed, the result was good in over two thirds of the cases

Tensor Fasciae Syndrome Ober Fasciotomy

Case M G—Female Adm. September 1929 Age 48 yrs.

Patient complained of tenderness at the left posterior superior spine and left gluteal region, with radiation Ober and straight leg raising signs were positive. In this case no relief was obtained from conservative treatment or from lumbosacral or sacro-iliac fusion Finally, the tensor fasciotomy performed seven years later gave complete relief

Heyman Stripping Operation Gluteal Myofascial Syndrome

Case V R.—Female Adm August, 1946 Age 43 yrs.

Low back pain of ten months duration with tenderness over lateral aspect of right posterior superior spine. Radiation along posterior surface of the thigh and the lateral aspect of the leg and foot on the right side. Leg signs were positive on the right. Novocain injection gave relief for two hours. No relief from conservative treatment. She finally was subjected to a stripping operation of the right posterior superior spine with complete relief

VII. THE TREATMENT OF THE HERNIATED INTERVERTEBRAL DISC WITH SCIATIC RADIATION

A CONSERVATIVE

Is it always necessary to operate in the presence of a disc? Most observers will urge a trial period of conservative treatment, except where there are unmistakable neurological signs A herniating disc may recede into the intervertebral space and protrude again on exertion or change of position The remissions and exacerbations of the sciatic pain are caused by this alternating back and forward slipping It is the fixed disc which does not move but remains in situ that should be removed by laminectomy (Scott and Young,[**] Kuhns[**])

B OPERATIVE

1 Treatment of the herniated disc by decompression and removal

The decision to operate is reached on the basis of the long duration of the sciatic pain, its resistance to conservative measures and the presence of neurological signs The objective of the operation is not only the removal of the disc but also the obliteration of the disc space, so that no further recurrence of the herniation can take place According to investigations of Key,[n] the defect caused by the removal of the disc is closed by a thin superficial layer of fibrous tissue, leaving the deeper portions of the defect gaping The space should be curetted thoroughly, the more completely the intervertebral space is obliterated, the better is the guarantee against recurrence

2. Should the spine be stabilized after the disc is removed?

We believe that in all cases of an unstable lumbosacral junction, the *laminectomy should be followed by fusion. Attempts have been made to* stabilize the vertebral bodies from a transperitoneal approach (Lane and Moore[n]), through which the entire disc is removed, and the empty space is packed with bone chips The operation is too formidable to gain general *acceptance Lane and Moore[n] list 36 cases, of which 77 per cent were relieved.*

The usual method is the fusion of the posterior arches We prefer the use of cancellous bone taken from the crest of the os ilii to solid bone grafts. The combined method of laminectomy and fusion is especially recommended in patients with separate neural arches or spondylolisthesis (Ghormley, Love and Young[14]), in patients with arthritic changes of the spine, in the static type of backache, and in spines showing lesser congenital anomalies such as sacralization and tropism. Comparing the combined operation with the simple laminectomy, Barr and Jason[1] found that the laminectomy alone does not relieve the back symptoms to the same extent as it relieves radiation When the spine was fused 73 per cent of the patients were freed of their back symptoms, whereas only 52 per cent were relieved when a fusion was not performed Some surgeons even advise the fusion operation alone without decompression. Farrell and MacCracken[18] report such a series of cases with neurological *disc signs treated* by fusion alone Of 27 cases treated by spinal fusion alone, 83 per cent were relieved of sciatic pain of the same number of cases treated by laminectomy and fusion, also 83 per cent were relieved Startling as this may seem it is not *surprising when we consider the changes which* occur with the movement of the spine in some herniations It only shows how essential it is to distinguish between the movable disc with fluctuating symptoms and the fixed disc which produces permanent complaints

3 Pseudarthrosis following fusion operation

A certain number of cases develop pseudarthrosis in the fused areas, most frequently at the level of the sacrolumbar junction Cleveland, Bosworth and Thompson[8] observed it in not less than 20 per cent of their 594 cases. It is least

frequent when the fifth lumbar alone is bridged to the sacrum and more frequent when the fusion covers the third, fourth and fifth lumbar vertebrae This raises the question, how long should the patient be immobilized following the operation? After laminectomy alone, the patient may get up within one or two weeks Following the fusion operation, he should remain recumbent for not less than six weeks and, after leaving bed, he should wear a well fitting back brace for at least six months.

VIII STATISTICS

A LOW BACK PAIN WITH RADIATION, CONSERVATIVE TREATMENT

Cases of low back pain with radiation, in which the procaine test shows the radiation to be of reflex character, respond well to conservative treatment. In 117 cases of our series who had low back pain with radiation and a positive procaine test, 95 cases or 85 per cent were completely relieved

B LOW BACK PAIN, SURGICAL TREATMENT

Our own statistics show that the lumbosacral fusion in cases of low back pain not diagnosed as herniated disc gave satisfactory results in 86 per cent (DeGoes[8])

C LOW BACK PAIN WITH RADIATION, HERNIATED DISC

The operative removal of the disc was the treatment of choice except in cases of reversible discs Of 29 cases reviewed by Dr Hyndman, Dr Wolkin and the writer, 25 cases were completely free from pain after operation, four cases had persisting pain In a further group of 10 cases, eight were completely free and only two had persisting pain, a total of 39 cases of which 33 or 87 per cent were relieved

IX SUMMARY

1 In the management of the low back problem it is desirable to apply more refinement and precision to the diagnosis and to make the indications more selective and less sweeping, especially so far as surgical intervention is concerned.

2 Ligamentous structures under strain or stress require immobilization Muscular structures under stress assume a state of spastic contracture and require manipulation and stretching. The first consideration is to be given to the strain of the ligamentous structures

3 Only those cases in which the strained ligamentous and muscular structures are beyond natural repair require operative interference.

4 In all cases of back pain and sciatic radiation which show a "trigger" point and in which the diagnosis of a herniated disc cannot definitely be established, the procaine test should be used to distinguish between referred and reflex sciatic pain

5 The diagnosis of a herniated disc does not necessarily mean that the operation is unavoidable. In cases with short and infrequent episodes a conservative trial treatment is indicated while persistent sciatica or frequent and prolonged attacks suggest a completely ruptured and adherent disc and constitute an indication for its operative removal.

6 Removal of the disc alone without subsequent fusion operation is justified only in cases in which there is no localized backache and where the sciatic radiation is the principal symptom. In all cases in which the sciatic pain is associated with persistent backache, the combined operation of laminectomy and fusion should be performed

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Lecture II

ON IDIOPATHIC SCOLIOSIS

I ORIENTATION

SCOLIOSIS has been recognized as a clinical entity separate from inflammatory destructive lesions of the spine since the first half of the 19th century (Hare,[7] Lonsdale[14]). The scientific foundation of our clinical knowledge of this deformity was not laid, however, until decades later, around the turn of the century when the clinical observations were augmented by pathological research (Lorenz,[16] Schulthess,[21] Wullstein[26,27]). This step secured at least a sound morphological concept. It took another decade or so to bring forth some information of the mechanogenesis (Lovett[17]). Little had been established so far regarding the etiology of scoliosis. The question was rather lightly disposed of under the general and meaningless term of "spinal insufficiency."[7] In the meantime the treatment of this deformity remained disappointing and Lorenz himself considered it as practically incurable. Then came the introduction of the operative methods. This, it was hoped, was to be the stroke to solve the Gordian knot. What it meant for the treatment of the deformity is one side of the story, with which we shall deal in this lecture. The other side is that it practically set a halt to further investigations on the nature of this deformity and that scientific contributions concerning its pathogenesis have become distressingly scant. Now, when it it being realized that surgery offers a solution for only a small minority of cases, there is all reason to take up again the thread of investigation.

Idiopathic scoliosis is a deformity of the spine which develops from an apparently physiological state under the influence of gravity. This distinguishes it from the congenital scoliosis where *pathological condition exists from birth,* and from the paralytic scoliosis because here *a pathological event precedes* the deformity. The fact that the deformity develops under gravitational influences implies that it is selective, and this again means that certain predisposing factors must be at work. So far, these factors have become ascertained only tentatively. It is in this direction that our search must proceed if we are to understand the nature of deformity and if we are to devise a sound policy for its prevention and cure.

II. PATHOGENESIS

In morphological terms, scoliosis is a deviation of the human spine in two directions, lateral and rotatory, that is, in the frontal as well as in the horizontal plane.

Other deformities occur *either* in the frontal *or* in the sagittal plane alone

The dorsum rotundum in osteochondritis is the example of a deformity strictly in the sagittal plane, an anteroposterior deformity The so-called *sciatic scoliosis* in low back pain or in the herniated disc is a deviation strictly in the frontal plane. In contrast, the true *scoliosis is a deformity in both planes, a lateral as well as a rotatory deviation,* furthermore, deformations of the thorax and pelvis are superimposed upon the spinal deformity It is the *combination* of these components which characterizes scoliosis

Can such a deformity be produced experimentally? Wullstein[34] tried it by strapping animals in scoliotic position Others (Haas[9]) attempted to produce it by destruction of the epiphyseal plates of the vertebral bodies. Schwartzman and Merrill[23] obtained the deformity by unilaterally stripping or excising muscle groups None of these experiments parallels actual conditions in life They show only that a deformity can be produced by prolonged scoliotic position or by destruction of normal muscular equilibrium, but they do not include gravitational stresses which are essential in the idiopathic scoliosis.

How does the scoliotic deformity develop out of the normal symmetrical spine?

1 The frontal plane component

LATERAL DEVIATION

In the beginning there is simply an inclination of one segment of the spine against another At first, this inclination is within physiological limits and under voluntary control *It can be reversed* Soon, however, this inclination is no longer under voluntary control, it exceeds normal physiological ranges *It can no longer be reversed*

This initial inclinatory phase is more often observed in the paralytic than in the habitual or idiopathic type of scoliosis *In the latter type, the initial feature* is more often that of spinal collapse By this is meant a sort of telescoping in which the vertebral bodies have undergone a translatory side shift against each other No such translatory movement is possible under normal conditions it is prevented by the normal rigidity of the intervertebral disc. Consequently *any degree* of translatory displacement is *pathological*

2. The component in the transverse plane, the rotatory deformity

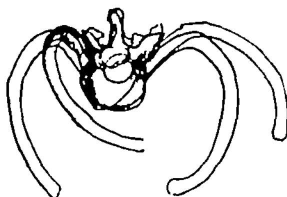
The vertebral bodies rotate against each other about a longitudinal axis. As the rotation exceeds the normal range of motion, it also becomes pathological and irreversible Moreover the rotatory forces transmitted to the structure of the vertebrae produce a torsion effect on the trabecular sysem, which changes their perpendicular course into a spiral one.

3 The change in the relationship between the spine and the thoracic cage

Under normal conditions the spine and thorax move as one piece in side bending and in rotation. In any position the spine divides the thorax and the

abdominal cavity in two symmetrical halves *In scoliosis* the situation is radically different, as the spine shifts sideways in translatory direction, it divides the thorax into two asymmetrical halves, the convex side being smaller than the concave side The same translatory shift of the lumbar spine divides the abdominal cavity into a smaller convex and a wider concave half In other words, the *scoliotic spine penetrates into the convex half of the thorax* and the convex half of the abdominal cavity

The essential and characteristic *feature in scoliosis* is that as the spine goes into side bending or rotation, *it does not take the thoracic cage with it* In other words cage and spine do not move in one unit, the thoracic cage stays behind on the concave side Similarly, in the lumbar spine, the column carries out a convex side rotatory movement but leaves the pelvis behind on the concave side (Farkas[3])


FIG. 64 Deformation of the thorax

Because the rib cage is anchored firmly to the thoracic spine through the costovertebral articulations, and the pelvis to the sacral spine through the sacro-iliac syndesmosis, *this disalignment necessarily results in certain deformations of the thoracic cage* and of the *pelvis* (Fig 64)

III THE PATHOLOGICAL CHANGES IN SCOLIOSIS

What morphological changes in the spinal column can be expected as the result of the two main components of the deformity, the lateral curve and the rotation?

A. THE VERTEBRAL BODIES

As a result of the lateral deformity, the height of the vertebral body is greater on the convex than on the concave side The vertebra is wedge shaped (Fig 65) Because of the greater pressure, the vertebral body becomes more drawn out convexly, but more condensed concavely As the curve increases, these inequalities become more marked In horizontal cross section the vertebral body is no longer oval but is now egg-shaped, the larger pole being on the convex side

The torsion of the vertebrae (Fig 66) The structural torque causes the perpendicular trajectories to assume a spiral course which can be demonstrated in the x-ray Externally it manifests itself by a peculiar twist between upper and lower surfaces of the body and a spiral shaped fluting of its surface That all these components—the translatory shift, the rotation and the torsion—are interrelated is shown by the fact that they all have a common maximal point. Rotation and torsion reach their highest level at the apex of the lateral curve

The transverse processes also change their direction They ascend on the con cave side and descend on the convex side of the body

B ARCHES AND PEDICLES

Marked changes are also seen in the column of the arches Their two halves no longer are symmetrical there is a kink at their attachment to the bodies and they appear thick and stubby on the concave and thin and drawn out on the



Fig. 65 Side bending, translatory shift and rotation deformation of thorax.

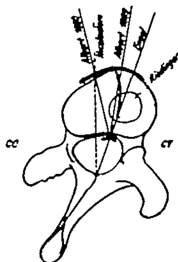


Fig. 66 Deformation of arches and pedicles and torsion of vertebra.

convex side The spinous processes shift sideways with the lateral deviation of the column although they do not truly parallel the curve, they are somewhat deflected toward the concave side so that the curve of the processes is flatter than that of the vertebral bodies The pedicles are shorter and stubbier on the concave than on the convex side (Fig 66) Also, they run in more sagittal direction on the convex side and they approach more the frontal plane on the concave side

C THE DISC

The key to the deformity is the pathological change in the intervertebral disc. Its significance for the development of habitual scoliosis has been pointed out already by Schulthess [31] The older observers believed that it plays only a passive role but we recognize today that deformations we see in the scoliotic spine would not be possible in the presence of a healthy intervertebral disc. The conclusion is inescapable that some degeneration of this structure is the primary factor which permits a subsequent translatory and rotatory deviation. The disc

is not only compressed on the concave side, but it also shows degenerative changes and fibrous substitution (Fig 67) The adjoining portions of the vertebrae become sclerotic, and there is reactive marginal lipping (Junghanns[13]) Aside from preserving the relationship of the vertebral bodies, the disc plays an important role in the so-called intrinsic equilibrium of the spine Only when it is destroyed by some degenerative changes and the resistance of this structure is greatly impaired can the described deformation of the spine take place

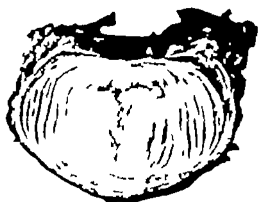


Fig 67 Degeneration of disc in scoliosis.

D THE INTERVERTEBRAL ARTICULATION

The obliquity of the vertebral bodies causes these articulations to converge toward the center of the curve so that the convex side articulations are elevated above the apex of the curve and are depressed below it. On the concave side, which is under increased pressure, the joints may become obliterated and fused together, and their reinforcing capsular ligaments may become ossified At the beginning of the deformity, when the spine is simply inclined laterally, the *tilted position of the articulations* acts as a sort of guide which directs the subsequent rotation of the vertebrae toward the convex side of the curve

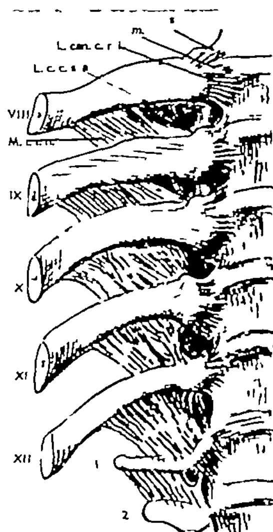


Fig. 68. The costovertebral articulations.

E THE COSTOVERTEBRAL ARTICULATIONS (Fig 68)

The articulations are deepened on the convex and flattened on the concave side The ribs are rolled up and exert pressure against the bodies and transverse processes on the convexity they are drawn out and flattened on the concavity of the curve

F THE THORAX

As the spine rotates independently without taking the thorax along, the ribs, anchored to the spinal column and spanned between the rotating spine and the non rotating thorax, must necessarily undergo some deformation

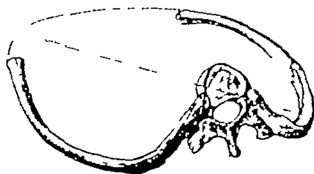


Fig. 69 The thoracic cage in scoliosis.

Posteriorly, the ribs are sharply angulated and curl up against the vertebrae on the convex side. They form a sharp prominence, the costal hump On the concave side the ribs are drawn out and the thorax is flat. In front the ribs are prominent on the concave and flat on the convex side (Fig. 69) The result of this distortion is that the long cross section diameter of the thorax is no longer transverse in the frontal plane but is now oblique, running from the convex side behind to the concave side in front.

G THE PELVIS (Fig. 70)

If the sacrum is included in the scoliosis, a deformation of the pelvis develops similar to that of the thoracic cage This inclusion of the sacrum in the curve depends entirely upon the configuration of the fifth lumbar vertebra, if its lower contour stands horizontal the lumbal curve ends there and *there is no pelvic tilt* If the lower contour of the fifth lumbar is oblique so that the lumbar spine is implanted obliquely into the pelvic ring then the fifth lumbar vertebra acts as the keystone of a curve which includes both lumbar spine and sacrum In this case the pelvis is tipped toward the side of convexity If both the upper and lower contours of the fifth lumbar are oblique but the wedge points in opposing direction, the fifth lumbar is not a *keystone* but rather a transitional vertebra which separates a lumbar curve from a sacral *counter curve.* The pelvis necessarily follows the sacral curve and is lower on the convex side of this curve. In



Fig. 70. The scoliotic pelvis.

respect to the lumbar contour curve the pelvic drop therefore appears on side of the *lumbar concavity*

Whenever one sees a pelvis tilting on the concave side of a lumbar cu instead of on the usual convex side (paradox scoliosis), one can be sure there is a lower sacral counter curve which determines the tilt of the pe and that the fifth lumbar is a transition vertebra between the lumbar and ral curves The deformations of the sacrum correspond to those of the tebrae The sacrum is compressed and stubby on the concave and longer thinner on the convex side In the longitudinal axis there is the same torqu seen in vertebral torsion If the sacrum is included in the lumbar curve, greater diameter of the pelvis runs from backward convex to forward cave, corresponding to the curve If there is sacral counter curve, the gre diameter of the pelvis corresponds to the latter Finally, *if the sacrum is included* either in a lumbar or in a sacral counter curve, that is, if both tours of the fifth lumbar are horizontal, *the position of the pelvis is nor* and there is no distortion

H. THE CHANGES IN THE SOFT STRUCTURES

Ligaments and muscles adapt themselves to the morphological changes of spinal column The anterior longitudinal ligament is thinned out and is displ toward the concave side The radiate ligaments of the costovertebral ar lations are elongated and thinned on the convex and thickened on the con side The muscles on the convex side show some atrophy and degenera Contractures are not uncommon on the concave side, and they often consti a formidable obstacle to correction, especially the lateral abdominal muscles the longissimus dorsi. In contrast, the large flat muscles, the trapezius, lat mus rhomboids and serratus, show very little change (H Virchow[23])

Secondary changes of the soft structures appear later, especially on the cave side of the curve, in the form of extensive ossification of the ligamen apparatus and synostosis between the arches and of the intervertebral artic tions *The ossification* of the intervertebral disc deserves special attentio seems to be more marked on the concave side while the convex portion of disc is still intact (Putschar[19])

Different from the true synostosis are the bridges and spurs due to a rea localized spondylarthritis They are particularly noticeable in the senes scoliosis of the lumbar spine (W Müller[16]) They protect the spine against ther collapse

Conclusions. 1 Muscle imbalance or muscular insufficiency alone can n account for the changes seen in *idiopathic scoliosis* No muscle action can duce a translatory shift or an excessive degree of rotatory or lateral disp ment Changes must occur in the non-contractile soft structures, the ligam and discs, which structures normally maintain the intrinsic equilibrium of spinal column and are the principal safeguards against deformation (see ture I)

2 As the deformity progresses, the secondary structural changes occur in bone as well as in soft tissues Structural changes in bone develop according to the general law of bone transformation, they are secondary to the changes in the soft structures The primary changes which lead to permanent and irreversible deformity are in the *ligaments and the disc*

IV THE CLINICAL ASPECTS OF HABITUAL SCOLIOSIS

A TYPES

According to the location, four types of curves can be distinguished (Schulthess[31])

1 The primary right dorsal curve

It is the most frequent In the course of development it pulls the trunk to the side of convexity, causing the so-called overhang The neck-shoulder line is

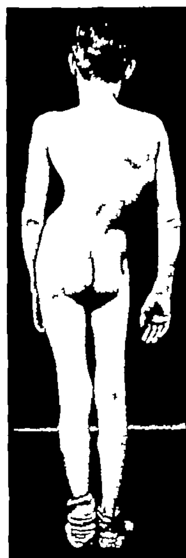


Fig 71 The primary dorsal curve.

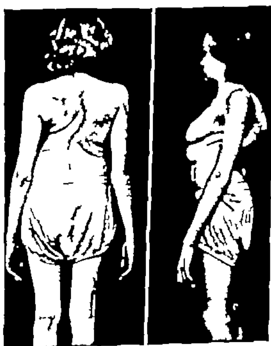


Fig. 72 The combined dorsolumbar curve.

asymmetrical, it is higher on the convex side As the body hangs over to the right, the so-called waist triangle formed by the loin, the iliac crest and the hanging arm is deepened on the concave and flattened and drawn out on the convex side The concave side hip protrudes and the shoulder girdle is no longer aligned squarely above the pelvis The rotatory deformity is marked by the convex side prominence of the ribs in the back and a corresponding concave side prominence in front (Fig 71)

2 Combined right dorsal and left lumbar curve

The combined right dorsal and left lumbar curve develops more often from a primary dorsal and less often from a left total curve The apex of the dorsal curve is the sixth to eighth dorsal vertebrae The elevation of the shoulder is on the side of the dorsal convexity, and the waist triangle is deepened on its concave side Types 1 and 2 comprise about 50 per cent of our cases of idiopathic scoliosis (Fig. 72)

3 Left total dorsolumbar curve

The left total dorsolumbar curve is generally the forerunner of the above mentioned combined dorsolumbar curve It represents therefore an early stage and is easily overlooked. We observed it in only 5 per cent of our cases (Fig 73) The apex of the curve is at the ninth or tenth dorsal vertebra

4 Primary lumbar curve

The primary lumbar curve is next in frequency to the right dorsal and the combined type We found it in 30 per cent of our cases The curve points to the left. It may end at the lumbosacral junction or involve the sacrum, in which case there is the above described pelvic tilt (Figs 74, 75)

5 Cervicodorsal curve

The cervicodorsal curve is unusual in idiopathic scoliosis Most of these curves belong to the congenital type

B THE DEVELOPMENTAL CYCLE OF THE IDIOPATHIC SCOLIOSIS

As one follows the progression of scoliosis, one cannot fail to recognize that nature attempts to bring the deformity to a halt. This attempt is directed toward recovery of the lost alignment between trunk and pelvis, i e , the posture and it consists in the establishment of neutralizing counter or compensatory curves In this sense we can accept the concept of self limitation of this deformity, in contrast to Lorenz, who believed that the deformity goes on irresistibly from bad to worse.



FIG. 73 The left total scoliosis.

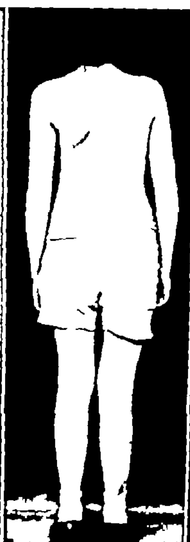


FIG. 74 The primary left lumbar scoliosis.



FIG. 75 Primary lumbar curve involving sacrum.

How often does nature succeed in developing these neutralizing counter curves? Our observations show that only in about 30 per cent of all types of idiopathic scoliosis a spontaneous compensation develops. Only to this extent is nature able to produce counter curves of sufficient length and angular value to restore the posture relationship between the trunk and pelvis In some types, as in the lumbar scoliosis natural compensation is frequent. In others, particularly the primary dorsal curve it is rare In the latter the spine stiffens early and extensively and no satisfactory counter curve can be established In contrast, the primary lumbar curve retains its mobility much longer, due largely to the effect of the gait upon the pelvis and the lumbar vertebrae

How long does the scoliosis progress? It is the general opinion that maturity sets a halt to the progress of the idiopathic scoliosis which means at the age of 17 or 18 years in boys and 15 or 16 years in girls. The best sign of maturity, according to Risser is the completion of the epiphysis of the iliac bone as it extends backward toward the sacro iliac angle.

V THE TREATMENT OF SCOLIOSIS

A THE GENERAL PRINCIPLES

The ideal objective of the treatment would be the anatomical correction of the curve, which entails the complete restoration of posture and the ability of the patient to maintain full correction by active muscle control A scoliosis is never completely corrected until the full mobility of the spine is restored and the curve can be reversed The question is, can this goal be achieved? Can one expect anatomic correction or at least a substantial improvement of the scoliotic curve, or should one be content with the restoration of the normal relationship between the trunk and pelvis, meaning restoration of posture? Also, what means are necessary to maintain either the improvement of the curve or the restoration of posture? This distinction is fundamental We find in practice that correction or even a substantial improvement of the scoliotic curve is impossible except in a small minority of incipient cases In contrast, if we set our goal at restoring the normal relationship between trunk and pelvis, we find that a large proportion of cases will respond to treatment by conservative means and that, moreover, the restored postural relationship can be maintained

B THE COMPENSATION TREATMENT (A Steindler[31])

1 What constitutes an adequate compensatory curve?

The recovery of posture involves the establishment of a counter curve equal in length and angular value to the primary curve Such a counter curve restores the normal relationship between trunk and pelvis It corrects the "overhang' of the trunk, the protrusion of the convex side shoulder or of the concave side pelvis

In order to establish a satisfactory lumbar compensatory curve both in lateral and rotatory direction the lumbar spine must be made movable by vigorous treatment and the back muscles must be trained to maintain control of the spine

The mobilization of the lumbar spine causes the lower end of the primary dorsal curve to be drawn into the area of the secondary curve, so that the primary becomes shorter and the secondary curve becomes longer To achieve a true compensatory

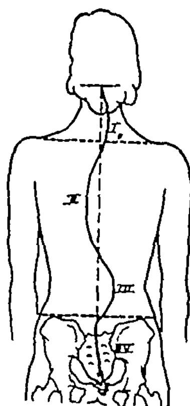


Fig 76 Compensation abolishes 1) Obliquity of shoulder line. 2) Elevation of convex side shoulder 3) Protrusion of concave side pelvis. A maximum of four curves is possible.

effect, it is necessary that the secondary curve be both of the same length and the same angular value as the primary Consequently, the success or failure of the procedure cannot be measured simply by the angular value of the primary curve alone because this is meaningless without its relation to the length and angular value of the compensatory secondary curve In other words, the angular value of the primary curve may be found decreased and yet the compensation has failed and *vice versa it may be increased and yet the compensation* is maintained It all depends on the mutual relationship of the two curves in length and intensity (Fig. 76)

2. How is compensation achieved?

The mobilization of the lumbar spine must be accomplished both in the lateral *and rotatory sense. Lateral mobilization is done by active and passive side bending exercises in standing sitting or lying down The rotatory mobilization* is accomplished by derotation exercises and maneuvers in sitting (the Grieve chair, Fig. 77), or recumbency on the derotation table (Fig. 78)

In derotation the *thorax must be directed toward the convex side and not the concave* (Fig 79) This is obvious since the thorax is already displaced toward the concavity Pushing the rib hump forward can only increase the disalignment between thorax and spinal column The passive manipulations are combined with active corrective exercises to increase the tone of the muscles and to teach the patient self-correction

3 How is correction maintained between exercises?

The compensation method aims as much at the correction of posture *by manipulation* as at maintaining this correction *by active muscle training* These efforts must be strictly correlated particularly must one avoid the error of mobilizing the spine in advance of adequate muscle development if one wants to avoid a collapse of the spine Pending this muscle re-education, it is necessary *either to keep the patient recumbent or to support the back intermittently by* supportive apparatus The object of the brace is to protect the muscles from fatigue and exhaustion

In practice it means that during the first period of the compensation treatment braces have to be worn for five or six hours a day this time is gradually diminished as the muscle development proceeds *Under no circumstances is a brace being worn continuously or indefinitely* (Fig. 80) The brace must be constructed on the three point principle The shoulder girdle, the scoliotic curve and the pelvic girdle This involves the anchoring of the brace against the trunk by a thigh piece which prevents tilting of the pelvis (Steindler[24]) In exceptional cases one may apply plaster casts. These must remain for short periods only, and on their removal the work of muscle development must be taken up again with all vigor *(Fig 81) A great deal of caution is required not to let mobilization* get ahead of muscle control.



Fig. 77 The Grieve derotation chair



Fig. 79 Correcting derotation toward the side of convexity



Fig. 78 The derotation table.

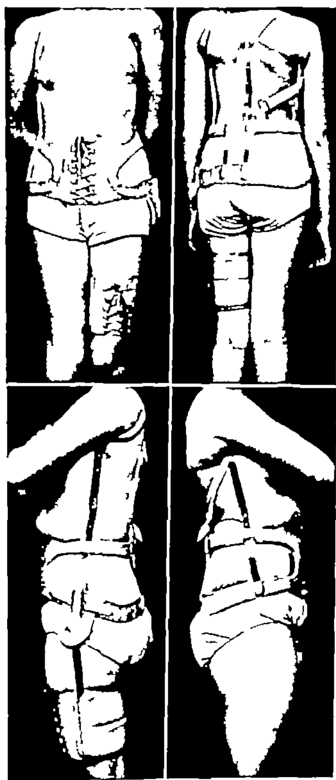


FIG. 80. Scoliosis brace.

What are the signs of adequate automatic muscular control?

a) If the x ray shows that both primary and secondary curves are equal in length and angular value, and if the overhang has disappeared

b) If the standing and recumbent length of the thorax are the same, as shown by the x-ray picture

c) If the extensibility of the spine is no more than normal

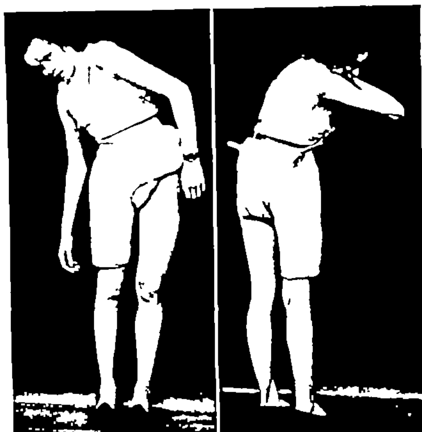


FIG. 81 Temporary casts.

d) If the normal sense of thoracic equilibrium is restored, that is, the patient feels that his corrected position is natural and not strained

e) If the posture is maintained actively without any visible effort

f) If the x ray shows by the completeness of the iliac epiphysis that maturity has been reached

5 What are the limitations of the compensation treatment?

Only 20 to 30 per cent of all types of idiopathic scoliosis acquire sufficient spontaneous compensation. The great majority of cases never reaches this state of natural compensation without corrective treament. Certain scoliotic types are susceptible to the compensation treatment, others are not

a) TYPES OF IDIOPATHIC SCOLIOSIS SUITABLE FOR COMPENSATION TREATMENT

1) The combined right dorsal left lumbar curve In this group it is possible to develop the lumbar curve to a state of adequate compensation and

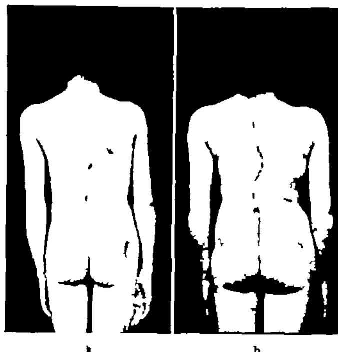


FIG. 82 Adequate compensation treatment primary right dorsal left lumbar combined curve. Case D B a) 1937 b) 1941

to maintain it permanently In a number of cases the angular value of both curves increases as the spine "settles" without loss of alignment. The percentage of good results with adequate and persistent compensation was 66 6 per cent (Figs 82, 83)

2) The primary lumbar curve. If the fifth lumbar is not oblique and the pelvis is not included in the curve, the results of conservative treatment are very good, adequate body realignment being obtained in 90 per cent of the cases (Fig 84) The obliquity of the pelvis complicates the situation A com

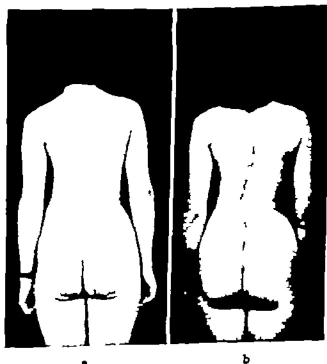


FIG. 83 Adequate compensation primary right dorsal left lumbar combined curve. Case B B a) 1936. b) 1939

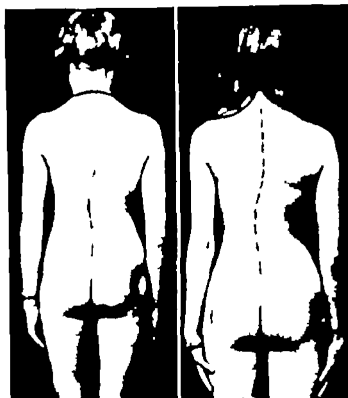


FIG. 84 Adequate compensation treatment primary lumbar curve Case B C. a) 1939 b) 1941

pensatory lumbosacral curve of an angle commensurate with the tilting angle of the pelvis must be secured to compensate for the pelvic obliquity We have often seen misguided attempts to strengthen the curve by Risser casts prior to fusion with disastrous results to the alignment between trunk and pelvis

3) The primary left dorsolumbar curve also responds to the conservative treatment. Here the percentage of successful compensation is as high as 75 per cent (Fig 85)

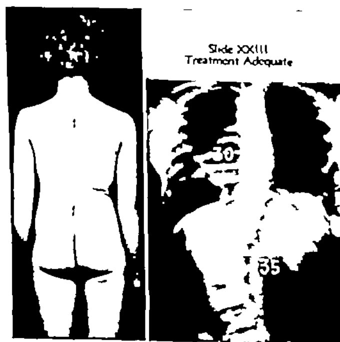


FIG. 85 Adequate compensation primary left dorsolumbar curve. Note equality in length and angular value of the compensatory dorsal curve

The Types Not Responding to the Compensation Treatment

1) The long and rigid primary dorsal curve

No satisfactory lumbar counter curve can be developed The muscles cannot control the posture in state of incomplete compensation and the spine breaks down, operative fusion becomes necessary (Fig 86)

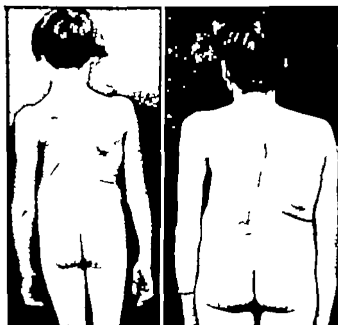


Fig. 86 Inadequate compensation long and rigid primary dorsal curve. Fusion becomes necessary Case M. O. a) 1931 b) 1942.



Fig. 87 Inadequate compensation obliquity of fifth lumbar Pelvic tilt not compensated

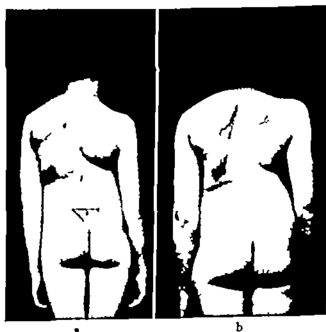


Fig 88. Inadequate compensation high dorsal curve. Fusion required. Case J B a) 1931. b) 1942.

2) The primary sacrolumbar curve with a wedged fifth lumbar and pelvic obliquity

Unless a secondary lumbosacral counter curve can be developed to neutralize the oblique implantation of the spine into the pelvis (see above), posture is not restored (Fig 87)

3) The high dorsal curve (Fig 88)

We have never been able to produce a satisfactory dorsal countercurve If progressive, these errors require operative fusion

C THE RESTORATION OF POSTURE BY CORRECTIVE CASTS

Because of the rigidity of the primary curve, all attempts of correction re sult in the development of counter curves Only in the early phases can there be a real straightening of the curve itself The earmark of anatomic correction is the reversibility, that is the changing of a curve into the opposite one This is possible in the mildest cases only We feel that corrective casts are justified as a part of the conservative treatment but only with certain reservations (Fig 81) First, the manner of applying the cast should harmonize with the pathomechanics of the deformity not only so far as the lateral curve is concerned, but derotation should be accomplished by pelvic rotation and the thorax should be adjusted to the spine by convex side rotation Secondly, casts should be applied only in long intervals and for short periods of time, two or three weeks, so as to have ample opportunity for muscle development and training

In applying corrective casts one should take into consideration the following points

1 All side bending efforts result in compensatory curves which may or may not be adequate

2 Slight forward flexion is advantageous in dorsal curves because of the "unlocking" of the intervertebral articulations (Lovett[17])

3 Derotation of the thorax must be *toward the convex* and not toward the concave side. The usually practiced rotation to the concave side is not rational because it only increases the disproportion between the thorax and spine, as already pointed out by Forbes[4] many years ago

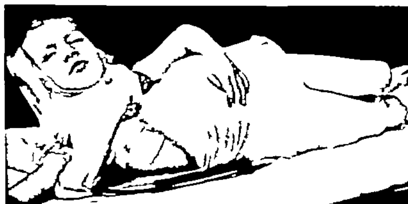
The method of Klein[14] also chooses forward flexion for the position of the corrective cast based upon Lovett's findings that in forward flexion the dorsal spine is unlocked and therefore allows rotation In the case of a right dorsal, left lumbar scoliosis the shoulders are rotated to the right and the pelvis to the left until the transverse axis of the pelvis and that of the trunk stand at right angles This confirms our view that derotation of the spine cannot be accomplished through the thorax but rather through the pelvis, as we do in our own derotation technique

D THE OPERATIVE TREATMENT

A wide field is left for surgical intervention by the limitations of the conserva tive treatment. We have cited certain types of cases in which the results of con-

servative compensation treatment have not been satisfactory Some of these cases cannot be improved by any treatment, some are undoubtedly benefited by surgery Only a proper selection of cases can establish the value of the surgical treatment of idiopathic scoliosis We note especially that such experts as Cobb, Von Lackum and DeForest Smith use a good deal of discretion in their choice of cases

FIG. 89 The Risser cast.



a



b



FIG. 90. The Blount extension brace.

1 The indications

The cases selected for operative fusion fall into four groups a) *The long right dorsal curve* in which there is no room for developing a satisfactory lumbar counter curve b) *the lumbar curve which includes the sacrum,* c) *the high dorsal and cervicodorsal curve* and d) *old scolioses* which "settle down past maturity and cause intercostal pain

There are cases of other types which may fail under conservative regime,

and there may be others in the groups mentioned as surgical material which make satisfactory progress under compensation regime In fact, it is difficult to make an operative indication at first glance, we usually observe the patient for one-half to one year under conservative treatment before making the decision In our series only about 10 per cent of all types of idiopathic scoliosis have come to fusion and, in the series of Cobb,[4] only 6 7 per cent

2. The technique

The preparatory step is to apply a corrective cast in order to obtain as much compensatory correction as possible before operation As long as the passive correction is to be followed by fusion, a good deal more corrective force may be used The most commonly used type of corrective cast is that of Risser (Fig 89) A good preoperative appliance is also the Blount[3] extension brace (Fig 90) The operation is carried out through an opening in the cast

Following Lange's use of wire and celluloid splints, Albee[1] used tibial graft, and Hibbs[8] introduced the local bone flap technique with the reaming out of the intervertebral articulation McKenzie Forbes[6] used the bone chip method and Schede,[30] the strut type of graft

What should be the extent of fusion? On this point the opinions are divided Some (DeForest Smith, Butte and Ferguson[22]) believe that all vertebrae in volved in the curve should be fused Others fuse only the apex region of the curve We believe that the fusion should always be extended beyond the limits of the pri mary curve In case of a double curve, it should include both the primary and secondary curves from the lowest completely horizontal vertebra above, to the highest below

E THE RESULTS OF FUSION OPERATION IN IDIOPATHIC SCOLIOSIS

The Research Committee of the American Orthopedic Association,[2] analyz ing over 200 cases of fusion of idiopathic scoliosis, found that some correction was obtained by the preoperative casts in 65 per cent of the cases, yet the average satisfactory end result after fusion was only 31 per cent, and only in 5 5 per cent was complete correction obtained There was also a considerable in cidence of pseudarthrosis reaching as high as 28 per cent.

In our own series all cases of idiopathic scoliosis which were operated on by fusion had been through a period of correction by compensation before op eration In some of these cases complete compensation was obtainable, and the operation was performed because of inability to maintain the correction In others, complete compensation was not obtainable, and the deformity was pro- gressing The results of the operations in these two groups are as follows

In the first group of 72 cases in which complete compensation was obtainable before the operation, correction was obtained and was successful in a high per centage. In the second group where fusion was performed without the preopera tive compensation, the deformity almost regularly relapsed, so that hardly any

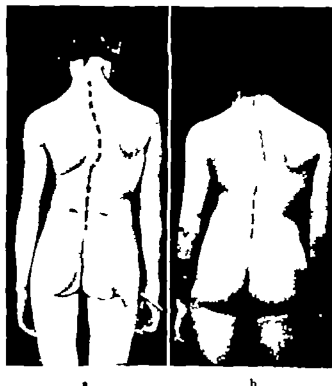


Fig. 91 Long right dorsal primary curve fair compensation. Holding after fusion operation observation seven years. Case J H. a) 1935 b) 1942

improvement over the initial condition was noticeable after five years or more (Figs 91 92)

The cosmetic operation of rib resection. Hoffa[10] in Germany and Hoke[11] in this country, and later Hoessli[12] in Switzerland introduced rib resection as a cosmetic operation to improve the posterior costal prominence. The operation

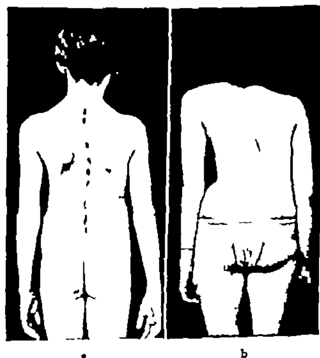


Fig 92 Failure of conservative treatment incomplete compensation. Fusion not holding observation eight years. Case D McK. a) 1936. b) 1944

can be performed in combination with the spinal fusion by using the resected portion of the rib as graft The rather sporadic reports indicate that the cosmetic result of this operation is satisfactory

F SUMMARY

1 The possibility of a natural arrest of the deformity by spontaneous compensation must be considered

2 Corrective efforts must be based upon the pathomechanics of the deformity One must recognize that the effect of lateral bending is the formation of counter curves and that alignment of the thorax to the spine by convex side rotation "unfolds" the concave side thorax

3 The effect of respiration on the thoracic spine must be appreciated, as must the effect of pelvic movements on the lumbar spine.

4 Forcible correction lessens the stability of the spine to a degree where it no longer can be neutralized by muscle action Cases undergoing this type of correction must be relegated to operative fusion

5 Operative fusion of the spine is not a method of choice but one of necessity It is reserved for cases which fail to respond to conservative treatment, either because compensatory correction cannot be achieved or because it cannot be maintained

6 For the control of a lumbar curve the important point is whether the pelvis is horizontal or oblique It is not possible to obtain realignment by compensatory curves in the presence of pelvic obliquity

7 Realignment by compensatory curves is important not only for the conservative treatment. It is also an essential prerequisite in cases undergoing fusion In cases fused without previous compensation the outlook for maintaining correction is poor

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Lecture III

ON THE INTERNAL DERANGEMENT OF THE KNEE

I INTRODUCTION AND PATHOGENESIS

A CONSTRUCTION AND FUNCTION

1 Joint construction

CONSIDERING the heavy static and dynamic demands placed upon it, th knee joint is, of all the large articulations of the body, the most exposed to, an the least protected against, mechanical insult It is the great shortener an lengthener as well as the main stabilizer of the extremity Its flexion-extensio range exceeds that of any other single joint in the body Its long lever arm pro vides its motions with speed as well as power

The joint has two degrees of freedom of motion, i.e, movement is possible i two planes perpendicular to each other flexion-extension and outward and in ward rotation. In this respect only it resembles the elbow joint. The latter, how ever, is a pure hinge with the articulations fitting closely together The kne joint is locked in complete extension against any rotatory movement, while i flexion the joint is loose so that rotatory motion between femur and tibia can b carried out.

To meet the requirement of unlocking the flexed joint for the rotatory mov ment which the mechanics of the gait demand, nature was compelled to fashic the contacting joint constituents in a very incongruous manner and to entru the stability of the joint entirely to muscular and ligamentous structures In i other large joint except the shoulder joint do we find such discrepancies in tl shape of the joint surfaces In anteroposterior direction the contours of tl femoral condyles represent a curve whose angular value constantly increases ai whose radius constantly decreases from front to back. The ratio of the radii the foremost and hindmost portions of the curve is 9 5 On the other hand, t opposing surfaces of the tibia are flat curves, and their radii are much larg than those of the curves of the femoral condyles The result of this incongrui is that the flexion-extension movement in the knee joint is not a pure hin motion At the beginning of flexion the movement is a pure rocking motion, that equidistant points of the femur touch in sequence equidistant points of t surface of the tibia After a flexion of 20 degrees, the type of motion changes now becomes a gliding motion The contours of the femoral condyles glide ove very circumscribed area of the tibial condyles, somewhat like a bow sweepi over a string This change is, however, not abrupt, and the motion starts wi true rocking, but soon the gliding element gradually increases and finally co pletely substitutes the rocking motion The joint is comparatively stable as lo

as the rocking type of motion prevails Past 20 degrees flexion the joint becomes gradually loose, and the reinforcing ligaments relax. The joint becomes free to carry out axial rotations This is quite in keeping with the mechanical demands of the gait. No weight is borne when the joint goes into flexion and it is then when in and outward rotation of the leg is carried out as an essential part of the gait during the swing and just before the take-off Besides with the joint being loose in flexion the wear and tear of the gliding motion on the tibial condyles is greatly diminished

2. Interposition

To make up for the incongruency of the constituents of the joint, there are interposed between them the semilunar cartilages and the fat pads The *inner semilunar cartilage* is C shaped, and its posterior half is closely fastened to the medial collateral ligament The anterior and posterior horns, however, are widely separated from each other in midjoint The anterior horn is attached to the anterior portion of the tibial articular surface near the anterior cruciate ligament Because of its shape and its attachment, the cartilage is rather stable and allows only little excursion.

In contrast, *the outer semilunar cartilage* is circular, that is, the anterior and posterior horns come closer together in the midline of the joint. It is less firmly anchored and has greater freedom of motion. Because of the natural valgity of the knee, it is the outer condyles of tibia and fibula which bear the greater amount of weight. As the length rotatory movements are performed, the outer condyle describes the greater arc, which it can do as it is freer the inner condyle is less detachable and therefore is in greater danger of being crushed or torn by this rotatory movement.

Interposed fat masses projecting under the ligamentum mucosa and the ligamentum alaria into the center of the joint fill out the remainder of the dead space between femoral and tibial condyles This protrusion changes with the movement of the knee and these masses depend upon the tension of the extensor apparatus to pull them clear of the condyles when the knee is extended.

Another protruding structure is the cruciate ligaments These ligaments and the projecting fat masses almost divide the joint into a medial and lateral half The cruciates play a considerable role in the stabilization of the articulation, they check motions of the knee The anterior and posterior cruciates have a length ratio of 5 3 but their insertion at the femur is much closer than at the tibia. The anterior comes from the lateral portion of the intercondyloid notch and goes to the anterior spine of the tibia the posterior cruciate ligament arises from the median portion of the notch and goes to the posterior spine The two ligaments cross each other and become tight in different positions of the joint. In extension the anterior is taut and the posterior relaxed almost to the finish of extension. Both ligaments, however, check backward and forward movements of the tibia against the femur

B WEAKNESS AND VULNERABILITY

1 External factors

There is a natural valgity of the knee due to the angle formed between the axes of the femur and tibia. The valgity places an undue stress upon the tibial collateral ligament This ligament is of considerable importance in the stabilization of the knee, and its posterior portion becomes taut in full extension The collateral ligaments also check abductory and adductory movements of the tibia against the femur and arrest rotation when the knee is in extended position In flexion the decreased radius of the sharper posterior curve of the femoral condyle permits the posterior portion of the collateral ligaments to relax

The valgity also produces an angular deflection between the extensor apparatus and the patellar tendon This deflection results in a lateral thrust exerted upon the patella when the knee goes into full exension There are certain natural safeguards to prevent the lateral dislocation of the patella It is held by the medial expansion of the capsular apparatus, and the forward projection of the outer condyle of the femur prevents the lateral displacement of the kneecap In addition the patella is held in place by the fibers of the vastus medialis which are inserted into the extensor apparatus lower than the lateral vastus

2. The intrinsic factors

The mobility of the knee is increased by the interposition of the semilunar cartilages The internal cartilage is loose at its anterior half, while the posterior half is woven firmly into the tibial collateral ligament This creates a danger zone between the movable and immovable portions of the cartilage which often responds to stresses by transverse tears The interposition of the medial and lateral fat pads also produces a precarious situation They depend for clearance from the impinging effect of the condyles entirely upon the tension of the quadriceps extensor apparatus This muscle is the only full extensor of the joint. The rectus femoris muscle is not able to accomplish full extension under ordinary conditions The latter is a function of the vasti Consequently any atrophy of the vasti will lead to weakness in ability to carry out full extension with force The strength of the quadriceps exceeds three times that of the combined hamstring muscles Its mechanical action is still increased by the insertion of the patella into the tendon as a sesamoid bone The contraction of this muscle not only tightens the tendinous insertion and puts the patella upward, but it also exercises a tension upon the anterior reinforcing apparatus of the knee and releases the fat pad

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II PATHOLOGICAL CONDITIONS INCREASING THE VULNERABILITY OF THE KNEE TO MECHANICAL STRESSES

A. EXTRINSIC

An *abnormal valgity* accentuates the stress upon the tibial collateral ligament. It facilitates lateral dislocation of the patella and increases the strain on the internal collateral ligament. Protracted strain on this structure produces chronic intermittent effusion which in turn leads to further distension of the collateral ligament and the posterior capsule of the joint. A pathological varus deformity places a strain on the fibular collateral ligament and greatly increases the pressure upon the internal semilunar cartilage One of the most common factors superinducing internal derangement is the hyper-extended knee. It is often the cause of early reactive osteoarthritic changes

B INTRINSIC

All chronic irritation whether due to traumatic causes or to other agencies produces hypertrophy of the synovial membrane and the fat pads These hypertrophies very frequently constitute a mechanical check to the extension of the joint There are also degenerative changes in the knee which may develop into mechanical obstacles and cause internal derangements of the knee, osteochondritis dissecans and osteochondromatosis of the knee belong to this group.

The knee is extremely prone to respond to mechanical irritation with chronic arthritic changes. *Here again the cartilaginous proliferations and exostoses may* often lead to production of spurs and the formation of free bodies in the joint which again will form mechanical obstacles

III PATHOLOGICAL REACTIONS OF THE KNEE TO TRAUMATISM

The manner in which the knee joint reacts to mechanical stresses is rather



FIG. 93 Case W A., with painful polyarthritic swellings of many years' duration. Pathological section from the knee joint reveals a hypertrophic villonodular synovitis. In addition, an arborizing lipoma and traumatic arthritis of the patella were observed.

typical and consistent Chronic stresses cause irritation of the synovia which results in effusion, either permanent or intermittent Acute mechanical stresses such as result in tears of ligaments or synovial membrane produce a joint hematoma In this event the swelling of the joint is rapid and intensive, and the high intra-articular tension prevents for a time the absorption of the bloody



FIG. 94 Case R. H (male) 28 years, with intermittent pain and swelling in knee joint for 18 months. Pain occurred only at night while at rest. Gross specimen of synovia very hypertrophic and mottled synovial villae with many foci of round cell infiltration. Note abundant intracellular hemosiderosis as well as multilayered synovial lining with large hyperchromatic nuclei.

contents The hematoma will remain liquid for a surprisingly long time if motion in the knee joint interferes with coagulation.

The synovial membrane reacts by thickening proliferation, and the production of villi and tabs (Fig 93) It is often of red or even chocolate color, and hemosiderin can be seen within the cells, its villi show abundant focal collections of lymphocytes (Fig 94)

Microscopically the tabs show marked chronic inflammatory changes with abundant plasma cells Secondary hyalinization of the fibrous tissues may occur and some tabs become avascular The tabs are particularly abundant in the so-called lipoma arborescens, a chronic non specific arthritis of the knee joint with greatly enlarged and traumatized fat pads After repeated traumatic events degenerative changes appear in the joint cartilage They follow the pattern seen in traumatic arthritis, which almost regularly follows repeated injuries to the joint. Defibrillated and eroded cartilage covers the articular surfaces of the femur tibia and patella (Fig 95) The x ray pictures show definite signs of degenerative arthritis (Fig 96)



FIG. 95 Traumatic arthritis Case M. D (female) #46-4519 46 years, May 1944 Six years before admission patient fell, landing on right knee had immediate pain and swelling which gradually subsided on rest. During subsequent years experienced 10 more episodes of trauma to same knee, each taking longer to subside. Examination showed knee diffusely enlarged and tender with marked crepitus on motion. X-ray (Fig. 96) showed arthritic changes. Failure of conservative treatment arthrotomy performed. Joint cartilage showed fibrillation and erosion fat pads hypertrophied and fibrotic. Finally an arthrodesis led to completely solid and painless, fused joint.



FIG. 96 X rays of case M D (Fig 95) showing changes of traumatic arthritis.

IV THE CLINICAL EXAMINATION OF THE KNEE JOINT

A HISTORY

The history reveals first the mechanical character of the original lesion sustained, secondly, the degree of injury, whether slight or severe, and thirdly, whether its effect was transient or persistent Symptoms produced by repeated injuries may be severe at first and become more and more attenuated or, on the other hand, the first episode may cause a slight symptom while the following injuries are followed by increasingly severer symptoms This is a point of diagnostic significance If the injury upon repetition of the trauma produces more marked symptoms, it is certain that a secondary reaction in the joint is in progress On the other hand, if the injury produces acute symptoms at first and lesser symptoms on repetition, this indicates that here is no progressive degenerative change in the joint but rather that there is a sort of biological adjustment taking place in the injured structures

B PHYSICAL EXAMINATION OF THE KNEE JOINT

1 Pressure points

The joint is more accessible than any other large joint in the body After taking cognizance of general swelling or infiltration, one can localize lesions of individual structures by distinct pressure points as follows A pressure point over the internal collateral ligament, a pressure point just in front of the latter for injured cartilages one next to the patellar tendon for a contused hypertrophied fat pad another one in the popliteal space for the distended posterior portions of the capsule, finally, a pressure line following the contours of the synovial attachment along the condyles of femur and tibia and circling the patella

2. Joint mobility

Intra articular pressure from effusion restrains flexion due to pressure of fluid against the anterior capsular apparatus Extension is limited by interposition of enlarged fat pads Forceful hyperextension strains the posterior capsule as well as the anterior cruciate ligament, which may rupture or become avulsed from the tibial tubercle Sudden restriction of the extension is called lockage It indicates impingement by a torn and displaced meniscus, by a fat pad or by a free body The lockage due to fat pad impingement is elastic and transitory A momentary lockage is caused by interposition of a free body Lockage in extension i.e , inability to flex, is seen in dislocation of the patella

3 Joint stability

Lateral stability is impaired as a result of relaxed or ruptured collateral ligaments Normally there is no lateral motion when the knee is fully extended and only a very slight degree of mobility in flexion Any considerable amount of

lateral motion is pathological, especially in extension. It is usually caused by a tear of the internal collateral ligament If the tear is in the superior portion of the internal collateral ligament above the cartilage, the latter follows the movement of the tibia If the rupture is in the inferior attachment of the ligament, then the synovial membrane and the ligament are sucked into the joint (Abbott, Saunders, Bost and Henderson[1]) If the collateral ligament is ruptured both above and below the cartilages, then the meniscus is loose The McMurray sign is elicited by flexing the knee and rotating the tibia inward It indicates lateral and rotatory instability

The *anteroposterior instability* or the so-called "drawer" sign means relaxed or ruptured cruciate ligaments It is the only abnormal movement which definitely indicates an injury to this structure. If the collateral ligaments are intact, the joint may still be stable in spite of the rupture of the anterior cruciate. Forceful posterior displacement of the tibia against the femur tears the posterior cruciate and causes avulsion of its tibial insertion

C AUSCULTATION OF THE KNEE JOINT

As early as 1885, Hueter tried to localize joint bodies by means of his so-called myodermato-osteophone The first American to use a stethoscope in examination of the joint was Blodgett, in 1902 C F Walters (1929), who examined 1600 joints by auscultation, pointed out that the number of joints giving auscultatory signs increases with age, from 1 5 per cent in the first decade to 81 5 per cent in the eighth decade The first graphic presentation of joint noises was described by Erb (1933), who recorded curves of the noises in sound knees as well as in knees with chondromalacia of the patella, with arthritis deformans and with meniscus lesions He used a so-called hard sound microphone sensitive to sounds transmitted by solid structures. For a number of years we have been interested in the auscultation of joints, particularly the knee (A Steindler[17])

An oscillogram is used for recording The graphic record is obtained by sending the sound waves through an amplifier They are then recorded, by means of a moving picture camera, on a running strip of paper on which time as well as the position of the joints is indicated on special lines In order to eliminate incidental noises produced by skin friction a double filter system is introduced to cut out all sounds under 130 and above 1100 oscillations To localize the origin of the sound the joint area is divided into lower inner lower outer, upper inner and upper outer quadrants The position of the joint in which the sound occurs is established by means of a goniometer The sound picture indicates pitch and intensity continuity or interruption. In this manner one can distinguish between crushing or crunching sounds which are fine low, weak and sustained grating sounds which are coarser, higher in pitch louder and sustained, cracking sounds which are harsh and sustained and isolated sounds which occur as cracks or pops or as low pitched thuds The latter are especially significant for the diagnosis of intra-articular mechanical derangement.

Normal sounds. In *children* under ten years the knee joint is uniformly

silent In *adolescents* it is silent except for an occasional fine grating in terminal positions. However, in *adults past middle age* one finds under normal conditions a more or less continuous soft grating accentuated in terminal position

Pathological sounds. In arthritis the sounds consist in grating and cracking in all quadrants and over the full range, accentuated in terminal position by isolated clicks or cracks *Osteochondromatosis* produces multiple sharp isolated cracks in one or more quadrants with an occasional sharp, high pitched grating In *osteochondritis dissecans* with a free body one hears an isolated click near full extension Auditory findings are particularly interesting in injuries of the *semilunar cartilage*, as they indicate location and type of lesion Particularly suggestive of a cartilage injury is a dull thud in the lower inner quadrant which appears either on completion of extension or flexion Not all similunar cartilage injuries give auditory signs They are missing in cases of soft and easily detachable tabs which float easily in and out in the intercondylar space In the majority of cases the auditory findings are of definite value In a series of 14 cases there was good correlation between the interpretation of preoperative sound recordings and the actual operative findings

V INJURIES AND DISEASES OF THE SEMILUNAR CARTILAGE

A TEARS OF THE SEMILUNAR CARTILAGE

Considering the fact that injuries of the semilunar are produced commonly by rotatory twist, one should expect longitudinal tears, partial or complete, to be more frequent than transverse fractures Actually, the majority of tears occurs in the anterior attachment, and the tears are incomplete

If the peripheral attachment is torn but the anterior intercondyloid attachment remains then displacement is not marked. Again, if the anterior horn remains attached by the transverse ligaments, while the peripheral portion is ruptured together with the posterior attachment, then a pure forward dislocation of the posterior end of the cartilage may occur Locking is caused not so much by a dislocated anterior attachment as it is by the more extensive displacement of the middle portion of the cartilage, such as occurs in the so-called bucket handle type

1 Tear of anterior attachment

In our experience it is more frequent than any other type of tear (Fig 97), though some observers (Enslin and du Toit[1]) believe the bucket handle tear to be more common The anterior tear has its tender point immediately behind the medical fat pad, and the signs of lockage are not quite so drastic as in the bucket-handle fracture

Often an abnormal mobility of the semilunar cartilage without gross lesions occurs when the coronary and synovial attachments have been stretched over a long period of time by recurrent joint effusion In this case the cartilage should

be removed, because it is abnormally mobile, but one should also explore the joint for other pathological conditions (Fig 98)

2. Transverse tears

Transverse tears of the internal semilunar cartilage as well as the less frequent oblique tears are commonly situated at the point where the posterior half of the cartilage becomes attached to the tibial collateral ligament (Fig. 99) In long standing cases of transverse tear a false joint may be established which makes the anterior half of the cartilage abnormally movable even though both





Fig 98 Case M. M Illustrating an anterior tear of the medial meniscus and hypertrophy of the fat pad

Fig. 97 Tear of the anterior attachment. *Case E M. (male) #M 10921 35 years,* March, 1936 Pain and locking following an inward twist of his knee six weeks before pain accompanied by swelling and inability to extend knee, locked at 160 degrees. Marked tenderness at anterior aspect of medial meniscus. Arthrotomy showed medial meniscus torn from its anterior attachment, the torn portion being interposed between the articular surfaces. Patient regained full range of painless motion after operation.

anterior and posterior attachments are preserved Such a condition may also give rise to locking (Fig 100)

3 Longitudinal fractures

Longitudinal fractures involve a portion or the whole length of the cartilage. To this group belongs the so-called bucket handle fracture in which the central fragment is dislocated into the intercondylar notch This type of tear produces a marked locking and auscultatory signs in form of a soft click or thud are usually present so long as the locking exists The finding of local pressure tenderness is uncertain (Figs 101, 102)

4 Tears of the posterior attachment

The tears of the posterior attachment and posterior tabs are usually not as

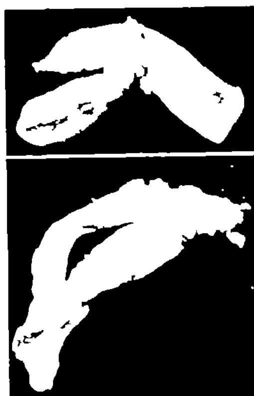




Fig. 99 (*upper left*) Transverse tear Case E Y (female) #33195 43 years, March 1936 Had three episodes of twisting injury within three years each followed by pain and swelling lasting two weeks. Right knee lacked 10 degrees full extension tenderness over medial meniscus. Arthrotomy revealed a transverse fracture of the medial semilunar cartilage Removal of the cartilage led to uneventful recovery

Fig. 100 (*right*) Anterior split with false joint. Case D M (male) #M-8357 18 years, April 1936. Patient complained of pain and momentary locking of right knee for two years following a baseball injury knee locked in 10 degrees flexion, with tenderness over anterior portion of medial meniscus. Arthrotomy showed medial meniscus with an anterior split and a false joint. Cartilage was removed and patient regained full and painless motion.

Fig. 101 (*lower left*) Bucket handle tear of medial meniscus. Case C. S #39-16306, 19 years, September 1941 Patient complained of pain and locking of right knee for one year after injury unable to fully extend knee and displayed an audible click at 135 degrees. Arthrotomy revealed typical bucket handle tear of medial meniscus removal brought complete relief



Fig. 102 Bucket handle tear Case S S #41 12151 24 years, September 1941 Patient complained of pain and locking of right knee of nine months duration after an injury Examination showed tenderness over medial meniscus and locking of knee at 150 degrees. Arthrotomy showed a bucket handle tear Removal of cartilage led to full range of motion

extensive as the tears of the anterior end The tabs float into the intercondyloid space and cause intermittent signs of internal derangement.

5 Tears of external semilunar cartilage

Tears of this cartilage are comparatively rare, because it is more movable and is less exposed to the grinding effect of rotatory twists The ratio is variously given as 1 20 to 1 4 Semilunar cartilage injuries of long standing are associated with chronic reactive joint changes There is hypertrophy of the synovial villi and intrapatellar fat pad The osteoarthritic changes of a traumatic arthritis may be expected in time, with formation of spurs and free joint bodies, a good reason for urging early operation on displaced semilunar cartilage.



FIG. 103 Cystic degeneration. Case W G., #41 12102 26 years, September 1941 Patient noticed gradually increasing swelling at lateral aspect of knee six weeks ago Examination showed cystic swelling at lateral aspect of left knee, tender only to deep pressure no limitation of motion. External semilunar cartilage was excised, and found to contain mass with cystic degeneration. Operation resulted in free, painless motion.

B CYSTS OF THE SEMILUNAR CARTILAGE

Cysts are ganglia originating between the peripheral surfaces of the external cartilage and the synovial membrane which covers it. They are most likely due to mucoid degeneration of connective tissue (Fig 103) There is still some controversy about the origin of these cysts and particularly why they are almost entirely confined to the external semilunar cartilage The symptoms of these cysts are pain which is not acute but rather dull in character tenderness to pressure and the presence of a palpable mass at the external cartilage (Figs 104) Their great prevalence in the external semilunar cartilage is striking I S McReynolds,[**] collecting over 257 cases, found the external semilunar cartilage

overwhelmingly frequent Of 170 cases reviewed by Bennett and Shaw, 143 were in the external and only 27 in the internal cartilage (Fig 105)

C THE DISCOID SEMILUNAR CARTILAGE (Fig 106)

This variation also occurs mostly in the external meniscus Marottoli,[17] studying cases in the literature and in his own clinic, found among 114 cases only two of a discord meniscus of the internal semilunar cartilage



FIG. 104 (*upper left*) Cyst of lateral meniscus of left knee Note swelling over head of fibula.
FIG. 105 (*upper right*) Cyst of medial meniscus. Case V M., #56-805 53 years, August, 1945 Patient developed an increasing lump at medial aspect of right knee following trauma 1½ years before. Operation revealed a cyst of the internal meniscus, 2½ x 1 inch in size this cystic tumor was filled with thick gelatinous material The microscopic slide showed a thick wall of connective tissue lined with one layer of connective tissue cells in form of an endothelial membrane



FIG. 106 Discoid semilunar cartilages.

The anomaly goes back to the persistence of the fetal character of the meniscus The annular form of the fibrocartilage is a secondary adaptation, and the disc represents an embryonic stage The surface of this meniscus is plain and smooth sometimes concave, sometimes convex and often it shows an elevation in the center The latter causes the so-called snapping or trigger knee There are also some changes in the external femoral condyle which is smaller on the

affected than on the sound side, its surface is often flat, the platycondylus of Putti Young of Glasgow was the first to describe this condition (1889)

Similar changes can be seen in the external plateau of the tibia, resulting in a considerable widening of the joint space between the external condlyes of femur and tibia

The symptoms of the discoid meniscus or the trigger knee are almost pathognomonic At a certain point the knee is caught by an obstacle and can complete the movement of extension only by a sudden jerk or jar occasionally occurring without any pain It is the central elevation of the discoid cartilage which produces the jerk. In addition there is an abnormal mobility of the disc itself

From the clinical point of view one may distinguish two types of trigger knee, namely the congenital and the traumatic. In the congenital type, again, there are two sub-groups There is the group which is discovered shortly after birth and where the trigger phenomenon does not constitute a real pathological process. There are no symptoms, and no particular treatment is needed In another more frequent congenital type the first symptoms appear in infancy or adolescence, not infrequently associated with, or precipitated by, trauma. Here pain and swelling may be present, although only to a slight degree in most cases This almost always indicates a partly loosened discord meniscus The clicking sensation is usually of greatest intensity just posterior to the lateral collateral ligament.



FIG. 107 Bilateral calcification of the lateral menisci. Case J H.

The traumatic cases of trigger knee are more frequent. Some show only the trigger phenomenon in others this symptom is combined with the signs of meniscus lesions

A relationship between the discoid meniscus and the cyst formation has been assumed (Kulowski and Rickett[14]) R Ollerenshaw[23] reports instances of cysts associated with malformations an argument for the congenital nature of the cyst Bilateral so-called giant menisci with mucoid degeneration are also reported by Herzmark[1] and of the 19 cases of cyst of the lateral meniscus which Kulowski[13] reports three were associated with variable degrees of malformation.

The treatment of the discoid cartilage depends upon the severity of the symptoms In the congenital discoid case where only the trigger point symptoms exist without functional disturbance no treatment is necessary In other cases, however where the symptoms are more severe and persistent and inflammatory reactions occur the treatment of choice is removal. In two cases reported by Marottoli[1] the discoid meniscus was removed and the symptoms subsided

D THE CALCIFICATION AND OSSIFICATION OF THE MENISCUS (J B Weaver[27])

Calcification of the meniscus can be demonstrated in the x ray picture and is usually found in the lateral meniscus The x ray shows a fine shadow which occupies the place of the lateral meniscus but has no bony structure (Fig 107)

VI THE CLINICAL PATHOLOGY OF SEMILUNAR CARTILAGE INJURIES

A SENSORY SIGNS

Local tenderness although significant is by no means constant, and it may be entirely absent between attacks The location of the pressure points is just lateral to the site of the fat pads A very useful sign is one described by A G Apley [2] The patient lies on his abdomen, and a preliminary check is made by rotating both knees externally and then flexing and extending the knees through a full range of motion to find at what angle the pain occurs The foot is then grasped with both hands, and after fixing the femur with the surgeon s knee on the back of the thigh, the patient s knee is distracted by means of traction and then externally rotated Pain produced by this maneuver is thought to be due to ligamentous injury rather than to the meniscus Pain on compression of the knee joint carried out in the same position indicates a meniscus injury

McMurray's sign consists in flexing and abducting the knee and then rotating it inward and outward In case of a loose or fractured meniscus the test elicits either clicks or pain sensation during the process of flexion and extension

B LOCKAGE

Restriction of motion by lockage particularly of full extension indicates that the cartilage is not yet reduced and that a reduction obstacle still eixsts Locking is a common sign, but it is not universal in injuries to the semilunar cartilage It is significant, however if it does exist There are some differences in the type of locking and one should ascertain whether it is absolute, soft, transitory or persistent. Longitudinal tears have usually a definite history of locking, and it is a rather constant sign in this type of tear (Ferguson and Thompson[8]) In tears of the anterior portion of the semilunar cartilage there is usually no history of locking but the patient states that he has a sensation of the knee giving way which is associated with pain and stiffness In tears of the middle portion of the cartilage the physical findings are usually the same

C ATROPHY

It is of the reflex type and involves mainly the quadriceps Atrophy occurs in all kinds of injuries of the knee joint It is especially acute in injuries to the semilunar cartilage where it is an early and very constant symptom

D THE AUSCULTATORY EVIDENCE

In the presentation of auscultatory signs in internal derangement of the knee it was stated that characteristic sounds appear almost regularly in injuries to the semilunar cartilage A moving meniscus gives a thud or a snapping sound. A grossly torn and frayed meniscus gives a crunching sound A high pitched click would indicate a bucket-handle fracture, and multiples splits, especially of the posterior part, give a succession of clicks When a wide bucket handle fracture is reduced, a loud crack and jolt is felt (Enslin and du Toit[7])

E X RAY VISUALIZATION OF MENISCAL LESIONS

Attempts have been made to visualize lesions due to internal derangement of the knee in the x ray picture The old method of injection of air or oxygen (Werndorf, Kleinberg) proved not very satisfactory Recently the question of the pneumo-arthrogram again came into the foreground, after Burman studied the possibilities of it for the knee joint. McGaw and Weckesser[10] report over 500 cases studied by pneumo-arthograms without serious complication Certain abnormalties can be detected by this method, for instance, irregularities in the contours of the meniscus, tears or shredding (Fig 108) Progress in visualization was made with the introduction of opaque media. Knute Lindblom[11] uses 9 cc of a 35 per cent solution of parabrodil Of 27 cases which he operated after an opaque medium x ray picture, he found his preoperative diagnosis verified in 25 cases



FIG. 108 Air arthrogram of normal knee.

VII. THE TREATMENT OF SEMILUNAR INJURY

The treatment for a ruptured semilunar cartilage is its removal. The approach to the cartilage depends upon the accuracy of the diagnosis If complications are suspected a broader utility incision is made The same incision is usually used for free bodies or fat pads If the lesion is more accurately localized a smaller incision can be used, either that of Robert Jones or that of Timbrell Fisher The latter incision has the advantage that it runs parallel to the outer margin of the internal collateral ligament and then curves forward toward the patella This gives easy access to the anterior portion of the semilunar but it is more difficult to reach the posterior portion. The advantage is that it avoids more of the sensory branches of the skin. *It is possible by this operation to remove all the cartilage or at least four fifths of it*

Following the operation there is a rapid and profound hypotonia and atrophy

of the rectus femoris as well as of the vasti Since the joint depends for its stability upon the tone of these muscles, their earliest rehabilitation is of prime importance For this reason, the early institution of active motion is necessary Most surgeons begin this on the second or third day, starting with patellar exercises and active extension (Moorhead and Lyal[11])

Should the entire cartilage be removed? Some surgeons feel that removal of all but the posterior portion of the cartilage would be sufficient and that the remainder can be left However, there are cases on record in which symptoms persisted when the cartilage was not entirely removed The posterior portion can be removed by the Henderson approach, through an incision running perpendicularly between the posterior edge of the condyle and the inner ham strings if it cannot be reached through the anterior incision

Should one trim or resect fat pads when removing the semilunar cartilage? Some slender adhesions of fat pads to the intercondyloid notch are often seen in cases which have repeated episodes resulting in hemorrhage and adhesions Adhesions should be severed, and all such tags or hypertrophied pads which are actually impinging should be removed We do not think that loose fat pads should be resected routinely unles they are definitely hypertrophic and threaten impingement.

Should the operation be done after the first dislocation, or should one wait for recurrences? If locking occurs and the patient is seen very soon afterward, then an attempt at reduction by the proper maneuvers should be made However, repeated attacks call for removal of the cartilage When reduction fails under anesthesia, the primary operation is urged by many authors (C R. Murray[22]), while others again prefer to wait until the sign of the immediate reaction has passed

What is the static effect of the removal of the cartilage? The joint space is narrowed at the site of the removed meniscus showing that the meniscus directly takes part in the supporting mechanism of the femoral condyle (Elfskind[6])

Does the meniscus regenerate? After partial meniscus resection part of the excised portion may be replaced by fibrous tissue ingrowth. The anterior portion joins the posterior part only at the peripheral zone, where it is supplied with blood This is an argument for the total meniscectomy rather than the partial one However, the total removal also may be followed by a replica of fibrous tissue On the whole though a re-organization of the meniscus is infrequent in man

STATISTICS ON REMOVAL OF THE SEMILUNAR CARTILAGE

In general, the statistics on this operation are very favorable Earlier reports (Surls and Osgood[29]) speak of 87 per cent good functional results for the internal and even better for the external semilunar cartilage. Recent statistics of MacElroy[14] covering a series of 1700 cases show 72 per cent complete cure and 18 per cent slight pain and weakness Our own statistics show good results

with no disturbance of weight bearing and no recurrence of locking or symptoms of any kind in 78.5 per cent.

VIII INJURIES TO THE CRUCIATES AND THE TIBIAL SPINE

As a rule a rupture of the cruciate ligament is associated with damage to the *internal collateral ligament, seldom is it found as an isolated rupture.* This rupture produces hyperextensibility and anteroposterior mobility of the joint and is regularly accompanied by a good deal of hemorrhage If the tibial spine is avulsed a bone block against extension is often established in the inter condyloid space.

A THE MECHANICS

The anterior cruciate ligament becomes tense in extension and hyperextension and relaxes with the beginning of flexion Together with the fibular collateral ligament, it therefore anchors the joint in extension The ligament checks inward rotation of the femur, abduction and particularly the anteroposterior movement of the leg.

The posterior ligament is relaxed in extension Since the accident usually occurs with the knee extended, the anterior cruciate is far more often injured than the posterior

B SIGNS AND SYMPTOMS

The diagnosis is easy in cases in which the x-ray shows the avulsion of the tibial spine. An abnormal anteroposterior mobility indicates interruption of the ligament Certain associated lesions must be differentiated (Bristow[4]) Direct injury to the tibial collateral or an abduction sprain causes tenderness and pain medially over the femoral condyle A rotation sprain that results in a torn cartilage with a torn coronary ligament causes lockage and inability to extend. Loose bodies also cause frequent though transitory lockings which do not occur in injuries of the cruciates Locking or "catching" is also seen in fat pads.

C THE TREATMENT OF THE RUPTURED CRUCIATE

The rupture does not always indicate surgery but proper immobilization is always necessary There are cases on record in which a perfectly stable limb was obtained by sufficient immobilization even though the cruciates had not been repaired This is particularly true in younger people It follows therefore, that conservative treatment has a definite place especially in acute cases. After immobilization in plaster a spontaneous repair of the ligament occurs in many cases In cases in which the relaxation persists especially the drawer sign, surgical repair of the anterior cruciate is indicated The question arises whether repair should aim at the reconstruction of the injured ligament, or whether the ligament should be substituted

Primary suture is difficult, particularly if it occurs close to the insertion. Most of the operations are based on the principle of substitution of the ruptured

cruciate ligament Two methods to be mentioned are those of Putti and Hey Groves[11] The Putti plan is the introduction of a strip of fascia lata into the joint to act as a cruciate ligament This is done by an oblique tunnel which goes through the femur and tibia in the direction of the ligament

The procedure of Hey Groves is similar except that he uses the tendons of the semitendinosus and gracilis inserting them into drill holes made in the internal condyle of the femur for reconstructing the posterior cruciate, while the anterior is fashioned from a piece of fascia

The method of Putti is the simpler one since it consists only in the insertion of a fascial strip in the direction of the anterior cruciate ligament through the joint from the outer condyle of the femur to the anterior spine of the tibia

IX FAT PAD IMPINGEMENT

The important diagnostic point is the tenderness to pressure at the site of the fat pad immediately adjacent to the patellar tendon Usually there is little pain and no limitation of flexion but full extension is limited As the tibia rotates outward at the end of extension the patient complains of pain There is absence of true locking although the patient has the sensation of the knee giving way In the beginning the enlargement of the fat pad is due to hyperemia In these cases immobilization and rest usually cause the edema and swelling to subside Later the fat pad becomes indurated and scarred and its changes are irreversible In this case the pad should be removed This is particularly so in the so-called lipoma aborescens found in women around the time of menopause

The treatment of fat pad impingement should start conservatively by immobilization and rest so as to give the fat tissue an opportunity to shrink Fat pads already indurated will persist in impinging during extension and will require operative removal

X. FREE BODIES IN THE JOINT

There are three sources of free bodies Either they arise from the synovial membrane by a process of metaplasia, or from osteoarthritic exostoses, or finally they represent sequestration of necrotic pieces of bone which fall into the joint as is the case in osteochondritis dissecans

A FREE BODIES OF SYNOVIAL ORIGIN

The osteochondromatosis

The pathological changes consist in a thickening of the synovial membrane, with the formation of cartilaginous bodies by metaplasia These bodies gain the surface and become detached from the synovia and then fall into the joint, sometimes in great numbers The symptoms are not well localized There is no tenderness at points diagnostic for injured cartilage or injured internal collateral ligament

As long as these bodies are still attached by a pedicle to the synovial membrane no serious or drastic symptoms occur It is different when they fall into

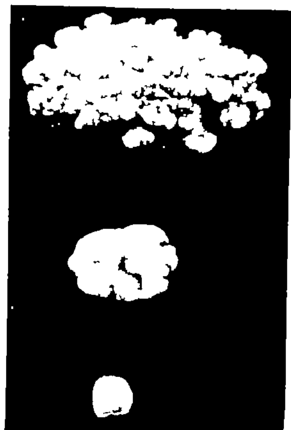


FIG. 109 Case D W (male) #46-3344 36 years, August, 1946. Patient, an achondroplastic dwarf with marked genu varum, complained of locking of left knee for six weeks. Examination showed the left knee locked at 145 degrees, with flexion possible to only 70 degrees. X ray revealed osteochondritic free bodies in both knees. Arthrotomy was performed on the left knee, and four large bodies were removed, one of which had been interposed between the articular surfaces. Uneventful convalescence one year follow-up.



FIG. 110. Osteochondromatosis. Case M. S., #38-10474 10 years, April, 1937 One month before admission repeated locking occurred in the left knee a large free body was palpable, and also visible in the x-ray Arthrotomy was performed, and several large free bodies were found. The synovial membrane, which was greatly thickened, was removed together with the medial meniscus this resulted in full range of motion. However locking recurred 1½ years later X-ray again showed formation of free bodies. A second arthrotomy was done, with removal of five more cartilaginous bodies. Patient remained symptom-free until five years later when signs and symptoms of osteochondromatosis reappeared x-ray revealed additional loose bodies.

the joint because many of them may conglomerate and form loose bodies of considerable size This may cause sudden attacks of severe pain followed by milder pain and momentary locking. Sometimes these bodies attain considerable size and become too large to be impinged between the joint ends. In this case they are palpable at the knee joint, particularly around the suprapatellar pouch (Figs 109 110)

B FREE BODIES ARISING FROM THE JOINT CARTILAGE

The osteochondritis dissecans

Osteochondritis dissecans is a circumscribed necrosis of subcortical bone usually situated at the lateral surface of the internal condyle of the femur In



FIG. 111 Osteochondritis dissecans. The joint body was attached to the synovia by a pedicle which carried the blood supply

the living bone surrounding the necrotic wedge a fibrous transformation of bone marrow occurs which ultimately separates the dead bone From the clinical point of view three groups can be distinguished (A G T Fisher[*])

1 A portion of the articular surface is completely detached and wanders about freely Then after an interval it becomes secondarily attached to the synovial membrane An injury is followed immediately by classical symptoms of locking which disappear after an interval (Fig 111)

2 A portion of the articular surface is completely detached, but it becomes almost immediately adherent to the synovial membrane Then



FIG. 112 Case O K. (male) #M 8197 20 years, February 1936 Patient injured his left knee 18 months before, playing football. Since then has complained of repeated sensation of momentary pinching in left knee. Arthrotomy revealed large, fibrosed fat pads in addition to a split of the medial meniscus in its anterior aspect also three osteochondritic bodies were found in the joint Following surgery patient was completely relieved.

after an interval it becomes detached again Here no classical symptoms occur after the injury, but they appear after an interval (Fig 112)

3 The detachment of the bone from the articular surface is gradual, and finally the body becomes free and movable In this case the process of gradual



FIG. 113 Osteochondritis dissecans complete detachment. Case F J

detachment is accompanied by attacks of pain and swelling which often increase in severity These are then followed by classical symptoms when the detachment is complete (Fig 113) In bodies which are completely free in the joint the superficial hyaline or fibrocartilaginous layers are formed by living cells, but the center is necrotic or calcified. The free body covered by living cartilage shows many indentations and irregularities (Fig 114)



FIG. 114 Osteochondritis dissecans. Free body of Case R. D

The x ray picture shows the defects in the internal condyle, often with a free body lying between the condyles in the intercondyloid notch (Fig 115) As long as the body still lies in its bed and is not completely separated, its sides are surrounded by a border of translucent tissue which represents the absorption zone of the living cancellous bone which is filled by fibrous bone marrow In incompletely separated bodies still attached to the matrix by a stalk or pedicle, the bone trabeculation can still be made out.

C FREE BODIES IN THE KNEE JOINT FROM OSTEOARTHRITIS

A third source of free bodies is osteoarthritic spurs They often break off from their base and produce free bodies which may cause some internal de-

rangement of the knee The symptoms are a sudden giving way of the joint, with a feeling of weakness and momentary signs of locking Large bodies usually lodge themselves outside the condylar plane and remain entirely harmless in the suprapatellar pouch or in the posterior compartment of the knee joint Smaller bodies, however, easily get caught between the condyles and then cause the fleeting lockage, associated with subsequent joint reaction Because of their effect upon the articulation as a whole, they should be removed



Fig. 115 Osteochondritis dissecans. X ray of Case F J (Fig. 113)

D LOOSE BODIES IN THE KNEE JOINT ARISING FROM THE MENISCI

Occasionally a fragment of the meniscus may become completely detached and remain in the joint as a loose body although these bodies are not of osseous nature (E D W Hauser[10]) Sometimes these loose bodies come from the posterior part of the meniscus or from a bucket handle fracture which is torn loose at the anterior end so that the bucket handle portion is forced posteriorly into the joint where it is caught between the articular surfaces It is gradually ground down and worn thin at the edge, forming a loose body Sometimes these menisci become calcified, and pieces will break off and appear as calcified free bodies

XI. THE INJURY TO THE COLLATERAL LIGAMENT

A. THE ANATOMY OF THE COLLATERAL LIGAMENTS OF THE KNEF (Brantigan and Voshell[3])

The tibial collateral ligament consists of an anterior parallel and a posterior oblique portion It is separated from the tibia by loose areolar tissue and by various bursae The anterior portion can be separated from the other joint structures, but the posterior portion blends with the fascia covering the popliteus muscle The anterior portion inserts into the epicondyle of the femur in

a fan shaped expansion The oblique portion, however, blends intimately with the capsule of the joint so that it cannot be separated from it.

In complete extension both the parallel and the oblique fibers are taut, and they restrain hyperextension In flexion the parallel anterior fibers become taut, while the oblique fibers are relaxed Also, in extension the oblique fibers restrain the posterior gliding but not the anterior The parallel portion again does not restrain either anterior or posterior gliding, either in extension or flexion. Together with the posterior cruciate the tibial collateral ligament restrains external rotation of the tibia upon the femur Abbott, Saunders, Bost and Henderson[1] also find that the anterior and superior portion becomes tight in flexion and checks abduction and adduction, but it relaxes if the knee goes into extension. At this position the posterior portion of the collateral ligament becomes tense.

The *fibular collateral ligament* is attached to the lateral epicondyle of the femur and is separated from the joint by the biceps tendon and a bursa The tendon of the popliteus coming from the lateral femoral condyle runs underneath this ligament. *The fibular collateral ligament is relaxed in all positions* of flexion, and it tightens with the beginning of extension, at 150 degrees. Injuries to the fibular collateral ligament are rare They usually occur in adduction flexion and inward rotation of the leg There is often an associated injury of the peroneal nerve as well as of the lateral head of the gastrocnemius or the iliotibial band.

Injuries to the tibial collateral ligament produce a tender point at its insertion or over the joint. Because of the tension of the posterior portion, complete extension of the knee is resisted. Forced abduction which separates the joint surfaces causes pain If the rupture of the tibial collateral ligament is associated with synovial tear, an hemarthrosis develops

Injuries of the collateral ligaments occur often in combination Not infrequent is the combination of tibial collateral, anterior cruciate and medial meniscus injury A strong blow on the upper tibia may rupture both anterior and posterior cruciates, but if both cruciates are torn and the collaterals are still intact, the joint is still stable in extended position. In flexed position however, the characteristic anteroposterior or drawer movement appears If the tibial collateral ligament is ruptured, as well as the cruciates, then this drawer movement becomes excessive

B TREATMENT OF THE RUPTURED COLLATERAL LIGAMENT

Severe sprains or ruptures of the internal collateral ligament are most disabling The pain is often very severe in most cases swelling and joint effusion occur after a short interval particularly if the synovial membrane has been injured directly Recurrences are frequent. They may be followed by hypermobility in extended position and the joint may become permanently unstable Significant is the absence of locking, but there is a great deal of wasting of the quadriceps In acute and subacute cases the best treatment is conservative. Immobilization of the limb in a plaster cast extensive enough to immobilize the

joint is the best measure for immediate relief, it is almost uniformly successful in the more acute cases

Cases which do not respond to this conservative treatment constitute only a small percentage of the acute ruptures In these, operative reconstruction is necessary This is often the case in older individuals or in those who have suffered from repeated injury, in which the relaxation becomes chronic For a chronically relaxed internal collateral ligament the procedure of Mauck[18] is to be recommended for its simplicity It consists simply in the transplantation of the ligament to a distal point where it is inserted at the internal condyle of the tibia, under tension This can easily be accomplished by a short incision at the medial aspect of the joint, reaching from the adductor tubercle to about 4 inches below the articular surface of the tibia

On the other hand, if the collateral ligament is completely destroyed, it must be reconstructed Here the method of Edwards[8] is to be recommended The plan of this operation is to make use of the tendons of the gracilis and semitendinous, which are severed above the knee joint and then strongly pulled up to be implanted into the internal condyle of the femur Instead of the tendons, a strip of fascia can be used with a distal base to be inserted at the condyle of the femur under an osteoperiosteal flap

In the rupture of the external fibular collateral ligament the method of Edwards[8] consists in the implantation of fascia lata into the fibula, and of the biceps into the lateral condyle of the femur

XII PELLEGRINI-STIEDA'S DISEASE[21]

This is an ossification or calcification of the internal collateral ligament due to trauma The characteristic feature is the deposition of calcium or of new bone in the region of the medial femoral condyle Early symptoms are those of synovitis of the knee with pain and swelling and limitation of motion which is gradu



Fig. 116 Pellegrini Stieda disease Case G B (male) #G-8342 29 years, December 1932 Patient injured his left knee three months before, while playing ball swelling and continuous pain at inner side of knee followed No locking but some limitation of flexion and extension. X ray showed calcified material in the soft tissues adjacent to the medial and posterior aspects of the left femur An incision was made over the medial condyle of the femur exposing a bony mass that infiltrated into the tendinous structures of the adductor magnus, in intimate contact with the internal collateral ligament. This tumor was not connected with the periosteum of the femur but arose in the soft structures of the region after removal the patient was free of pain.

ally progressive Examination shows local tenderness over the medial aspect of the femur with an enlargement.

The treatment consists in immobilization in a splint or cast. Surgical removal of the ossified ligament is seldom necessary[11] (Fig 116)

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Lecture IV

ON STATIC DEFORMITIES OF THE FOOT AND ANKLE

I ORIENTATION

LIKE all so-called static disorders those of foot and ankle cannot be explained entirely as being caused by external forces We must assume that other factors are at work, either constitutional or acquired, which lessen or impair the normal resistance against static stresses The study of the static ailments of foot and ankle therefore resolves itself, on one hand, into investigating the normal conditions for the static and dynamic equilibrium of the structures involved and, on the other hand, into the search for predisposing elements which may be of constitutional or acquired nature

The plan is to begin by investigating the normal weight bearing stresses and their distribution in the foot, first at rest and then in motion, considering in proper sequence all three principal articulations, the ankle, the subastragalar joint and the midtarsal joint On that basis we propose to investigate how the distribution of weight bearing stresses changes under pathological conditions, which include morphological phenomena and adaptive changes in the skeleton, muscles and ligamentous structures

II THE MECHANICS OF THE NORMAL FOOT

A. STATICS OF THE NORMAL FOOT

1 Construction

The mechanical stresses to which the foot is subjected can best be studied from the trajectory systems of the tarsal bones These systems represent the distribution of pressure and tension stresses Starting from the tibia one sees that the trabecular lines which receive the superincumbent weight from above converge toward the astragalus The center of this bone is the mechanical center of the weight bearing stresses

From here the trajectory lines stream backward toward the posterior process of the os calcis and forward through scaphoid cuboid and cuneiforms to the metatarsals In the sagittal sections we notice that in the planes of the fourth and fifth metatarsals the stress lines do not pass through the astragalus, but they pass from a dense sclerotic area at the upper surface of the os calcis forward to the cuboid and into the two outer metatarsals and backward toward the heel On the medial side however the stress lines pass from the body of the astragalus forward through the head and neck of the astragalus to the three cuneiforms and the three inner metatarsals and then backward into the heel (Fig 117)

This divides the tarsus structurally into two systems a medial one having its center in the body of the astragalus and a lateral one with its center in the os

calcis These two systems diverge in forward direction, one streaming into the three medial and the other into the two lateral metatarsals The two systems *represent the separate structures of the so-called inner and the outer longitudinal* arches of the foot The outer arch is the foundation, the inner rests upon the outer with the astragalus, being borne by the sustentaculum The os calcis is the common posterior support of both arches Anteriorly the arches separate the inner three metatarsal heads, serving as anterior support for the inner arch,

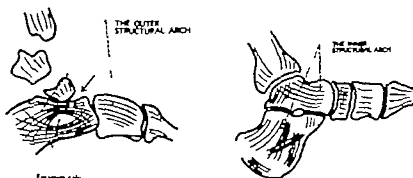


FIG. 117 The structural arches of the foot. (From A. Steindler *Mechanics of Locomotion*, Springfield, Ill Charles C Thomas, 1935 pp. 257 258, Figs. 4A and 4B)

while the outer two metatarsal heads are the supporting pillars for the outer arch (A Steindler[39])

2. Weight distribution in standing

According to D J Morton,[31] the weight distribution of the standing foot can be divided into three partial forces, namely a posterior portion, an anteromedial portion and an anterolateral portion The distribution of the weight between the calcaneous behind and the medial and lateral portions of the ball of the foot in front is 3 1 2 The mechanical axis between the outer and inner portions of the forefoot falls, in the ordinary standing between the first and second metatarsals in which case little weight is borne on the fifth metatarsal.

From the anatomical configuration of the arches as described, we see that for all practical purposes the posterior contact of the lower extremity with the floor is a circumscribed point Anteriorly, however, the contact is not a point but an oval shaped area, which takes in the heads of the metatarsals and their immediate surroundings Therefore while the weight bearing stresses find their reaction concentrated in the heel at one single point, in the forefoot the reaction is distributed over the entire ball of the foot.

3 Ligamentous reinforcements of the tarsus

The fact that the continuity of the skeletal structures is interrupted by three articulations makes it difficult to maintain the normal relationship between the bones of the tarsus when the foot is under stress The relation of the axes of these three major articulations of the foot is as follows In the ankle joint the axis runs obliquely to the axis of the knee joint, namely from outward and back

ward to forward and inward so that it does not stand quite in the frontal plane like that of the knee joint When the patella looks straight forward, the axis of the ankle joint is deflected as indicated The joint has one degree of freedom of motion, namely flexion and extension The subastragalar joint has only one degree of freedom of motion, namely pro and supination The axis of this joint is not strictly sagittal, but it runs from backward and slightly outward to forward and slightly upward and inward This deviation from the strictly sagittal plane corresponds to the deflection of the inner arch against the outer arch, already mentioned as being 25 degrees

The three axes of the midtarsal joint, which has three degrees of freedom of motion, correspond more nearly to the cardinal planes of the body The axis for pro- and supination is strictly sagittal, that for ab- and adduction is truly vertical and that for dorsi and plantar flexion is approximately frontal

The integrity of the foot is entrusted in the first place to the ligaments and then to the muscular apparatus The *astragalotibial* articulation is reinforced by internal and external deltoid ligaments The *subastragaloid* articulation is secured by the powerful calcaneoscaphoid or spring ligament which actually forms a part of the articulation By supporting the head of the astragalus from below, this ligament prevents the astragalus from gliding forward under the superincumbent body weight.

There are numerous smaller ligaments which reinforce the articulations, such as the calcaneocuboid ligament or ligamentum plantare longum which secures the calcaneocuboid articulation from below The heads of the metatarsals are held together by the intermetatarsal ligaments In contrast to the posterior part of the foot which is fairly rigid the mobility increases in forward direction, the toes and metatarsals showing the greatest freedom of motion

The midtarsal joints which permit motion in pro- and supination as well as in ab- and adduction make allowance for the adaptation of the toes to the floor The mobility of the heads of the metatarsals against each other further facilitates the adaptation of the ball of the foot to the contacting surface A free play of the toes is especially valuable for the gripping action of the foot in walking.

B DYNAMICS OF THE NORMAL FOOT

1 The articulations

The system of two arches supporting the foot is based on the maintenance of balance at rest and during locomotion A joint is established where the inner arch rests upon the outer arch The subastragalar joint The object of this articulation is movement between the arches about the longitudinal axis of the foot Pronation and supination So far as the purely static function of the foot is concerned that is the maintenance of equilibrium at rest such a joint would be of secondary importance In standing, balance is maintained in the sagittal plane that is in forward and backward direction, by the ankle joint which can adjust the line of gravity so that it always falls within the area of support,

between ball and heel In the frontal plane, that is, in side swaying motion, balance is maintained by the subastragalar joint This articulation adjusts the line of gravity coming through the astragalus so that it also falls within the supporting surface which is, when standing on one foot, the contact surface of the sole of the foot.

The meaning of this articular connection between the two arches becomes more apparent in the dynamic function of the foot, specifically in the mechanism of propulsion Propulsion is carried out by the action of the big toe at the moment of take-off This is an effort in the direction of pronation Because the incumbent weight of the body fixes the big toe to the floor, no actual pronatory twist is carried out, but the toe is held in equilibrium by the reaction of the floor against the ball of the foot Complete fixation does not occur until the full weight of the body has become shifted upon the ball of the foot toward the big toe side. The forepart of the foot digs into the ground with a gripping movement of the toes which have to adapt themselves closely to the unevenness of the ground. Therefore, a certain movement between the inner and outer arches is necessary *as the weight shifts during the gait from the outer to the inner border of* the foot It is for this reason mainly that the subastragalar joint is established

Supplementing this joint is an auxiliary articulation established in front of it this articulation is formed by the os calcis and cuboid at the outer side and the head of the astragalus and scaphoid at the inner half of the foot It is the mid tarsal or Chopart s joint. This articulation has three degrees of freedom of motion About a sagittal axis it has a slight pro- and supinatory range which supplements the pro- and supination movement in the subastragalar joint. In *the perpendicular axis a slight degree of ab- and adduction is possible in the* normal foot this range as well as pro- and supination may be greatly increased under pathological conditions The third movement is about the frontal axis, in the sense of flexion and extension of the forefoot against the back part of the foot. This movement while minimal under normal conditions is greatly increased in certain pathological situations, as clubfoot or flatfoot.

2. The active equilibrium. Myokinetics of the foot

The maintenance of active equilibrium of the foot at rest as well as in motion is the function of the muscles.

a) The Ankle Joint

The principal extensors of the foot are the tibialis anticus, the extensors of the toes and the peroneus tertius The principal flexors of the ankle joint are the triceps surae muscles and the lesser are the flexor muscles of the toes the peroneus longus and the tibialis posticus According to R Fick,[4] the total work which the extensors of the ankle are capable of amounts to 4.27 kgm., while the flexors are capable of 18 68 kgm this shows that the flexors of the ankle are at least four times as strong as the extensors, due to the fact that they include the powerful gastrocnemius and soleus.

b) The Subastragalar Joint

This joint is the principal articulation for pro- and supinatory motion Supination is carried out by the tibialis posticus, assisted by the flexor of the big toe, the common flexors of the toes and to a slight degree the tibialis anticus The total work capacity of the supinators is, according to Fick, 7 86 kgm Pronation is carried out by the peroneus longus and brevis and is further assisted by the common extensors of the toes and the peroneus tertius This group has a total work capacity of 3 22 kgm

c) The Midtarsal Joint

The only significant motion under normal conditions is pronation and supination The pronators are the peronei, the extensors of the toes, and the peroneus tertius, with a combined working strength of 1 45 kgm , while the tibialis anticus and posticus and the flexor of the big toe, as well as the flexors of the other toes are supinators with a combined strength of 1.39 kgm It is the tendo achillis, which when added to the supinatory muscle group is the definite factor which gives this group superiority When the foot goes into pronation we see that the tendo achillis is bowstrung so that when the foot is weighted down, it is forced by the short heel cord into a valgus position In other words, the short heel cord prevents the foot from going into supination and dorsiflexion at the same time, therefore, when dorsiflexion is imposed upon the foot by the superincumbent weight, the foot must automatically go into a valgus position

From experiments it is estimated that of the total tension stress developed by the muscles supporting the longitudinal arch, no more than 15 or 20 per cent is borne by the posterior tibial and peroneus longus The great portion of the tension stress is sustained by the plantar ligaments of the foot It is the function of both the inverting and everting muscles of the foot in their synergistic action to preserve a certain constancy in the weight distribution over the metatarsals at the ball of the foot Any deflection from the normal equilibrium which throws the weight line to the inner or the outer side will destroy the normal weight distribution

C THE ANALYSIS OF FORCES ACTING ON THE FOOT IN STATIC AND DYNAMIC CONDITIONS

Having become acquainted with the principal structures which are engaged in the static and dynamic functions of the foot, it is possible to analyze in mechanical terms the forces which maintain equilibrium in standing and walking

In walking the foot is placed on the ground, heel first It then rolls over to the outer border until the ball of the foot is reached From there on the weight line switches over to the inner side, while the toes dig themselves into the ground in order to provide a fixed point from which the body must be pulled forward As the heel then leaves the ground the deploy begins The knee is first flexed, with this flexion there is an associated inward rotation of the leg The foot is adducted in the midtarsal joint and a pronatory motion of the foot in the sub-

astragaloid joint follows The take-off is then completed by the flexory action of the big toe In order to give a clear conception of all these dynamic events, we try to present them in form of diagrams which illustrate the conditions of equilibrium in the different joints

1 The ankle joint

Let us suppose first that the line of gravity in upright standing falls through the center of motion of this joint, that is, in the center of the body of the astragalus In this case the gravital forces produce two counter reactions, at the ball and at the heel, which are the contact points with the floor These two reactions *are effective in translatory direction No rotary component acts upon the*

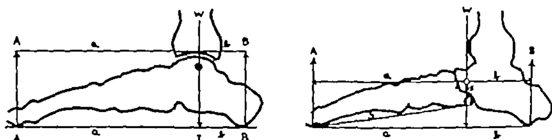


FIG. 118 (*upper left*) W = *Superincumbent weight. A = Anterior reaction. B =* Posterior reaction. I = Intersection point of line of gravity with base line. a = Distance of A from I. b = Distance of B from I. ○ = Center of ankle joint. Force analysis of ankle joint. Situation II. Sagittal plane equilibrium in ankle joint The line of gravity falls through the center ○ Condition of equilibrium Aa = −Bb. (From A. Steindler *Mechanics of Locomotion* Springfield, Ill., Charles C Thomas, 1935 p 271 Fig. 21)

FIG. 119 (*upper right*) S = Tension of the structure of the sole, s = Distance S to center ○. Force analysis in midtarsal joint. Situation V Sagittal plane equilibrium in midtarsal joint. Line of gravity through center ○. Condition of equilibrium Aa = −Ss. A = W b/a + b If a = O. A = W (maximum anterior reaction) and Wa = −Ss. (From A. Steindler *Mechanics of Locomotion* Springfield, Ill Charles C Thomas, 1935 p. 272 Fig. 24)

ankle joint itself, since it is assumed that the line of gravity falls through the center of motion (Fig 118)

Actually however the line of gravity does not fall through the center of the ankle joint in upright standing but, rather, at some distance in front of it, usually 4 cm In this event gravity develops a rotation component in respect to the ankle joint which tries to rotate the foot in the direction of dorsiflexion If this force is to be neutralized a rotation force will have to be developed of the converse order and of the same magnitude This force is supplied by the tension of the tendo achillis

By simply leaning forward we can cause the line of gravity to move still farther away from the center of motion in the ankle joint until it falls into the ball of the foot In this event the tension of the tendo achillis must be increased because the rotation moment which is produced by the reaction to the gravital

force is increased as the line of gravity wanders away from the center of motion Therefore, the tension of the tendo achillis which neutralizes this rotary force must be increased correspondingly

When by leaning forward the line of gravity has been thrown as far forward as the ball of the foot, the tension of the tendo achillis must be three times the body weight which rests upon the ball of the foot, because the distance of the ball of the foot from the center of the ankle joint is about three times as great as that from the ankle joint to the attachment of the tendo achillis

2. The midtarsal joint

We consider first the motion which occurs in the sagittal plane, i e., plantar

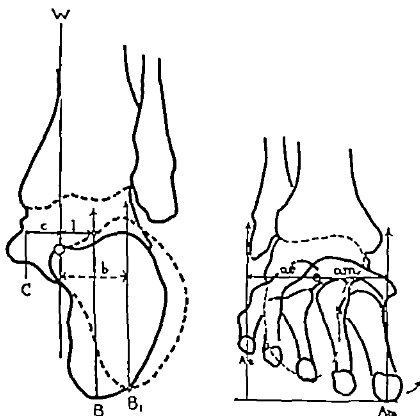


FIG. 120 (*upper left*) B = posterior reaction. 1 = Distance of posterior reaction from center O. C = Tension force of supporting structures at sustentaculum and internal malleolus. c = Distance of C from center O Force analysis in subastragalar joint Situation VII. Frontal plane. Equilibrium in subastragalar joint. Condition of equilibrium. Bb = −Cc. The greater the distance of B from center (pes valgus b) the greater must be C (c being equal) (From A. Steindler *Mechanics of Locomotion* Springfield Ill Charles C Thomas, 1935 p 274 Fig 25)

FIG. 121 (*upper right*) A = Total anterior reaction. Am = Total a.R. medially to center Ae = Total a.R. laterally to center A = (Am + Ae) O = Center of midtarsal joint for movement in frontal plane, pro- and supination. am = Distance from O. ae = Distance Ae from O Force analysis in Lisfranc's and Chopart's joints. Situation VII. Frontal plane anterior view Compensatory supination of forefoot. Effect of pronation of backfoot upon forefoot equilibrium in respect to midtarsal joint Condition of equilibrium. Am × am = Ae × ae i.e. The forefoot must go into supination until the moments of the medial reaction equal those of the lateral. (From A Steindler *Mechanics of Locomotion* Springfield, Ill., Charles C Thomas, 1935 p 274 Fig. 26)

and dorsiflexion or heightening or flattening of the arch. If the line of gravity falls through the center of this joint, the translatory effect of the reactions at ball and heel will force them straight upward, trying to flatten the arch This movement must be held in check by the structures of the sole of the foot, that is, the long and short flexors of the toes, the tibialis posticus and all the ligamentous structures of the sole Should the tension of the muscles of the sole of the foot as well as the ligaments be inadequate to resist the upward thrust of the gravital reactions, the result would be a forcing upward of the ball of the foot in the direction of dorsiflexion, and the foot would flatten out The cup-shaped ligaments would thereby be stretched, and the astragalus would be displaced downward, inward and forward In other words, the foot structure would experience a break at that point (Fig 119)

3 **The subastragalar articulation**

Motion occurs in this joint in a pronatory and supinatory direction. Projecting the joint in the frontal plane and looking at it from behind (Fig 120), we find that the line of gravity in upright standing falls outside of the supporting point of the heel therefore gravity has the tendency to force the foot in the direction of pronation This is counteracted by the resistance of the ligaments and the tension of the supinatory muscles.

Looking at the joint from in front and examining the situation of the metatarsal arch we find that the weight bearing stresses distributed among the metatarsal heads keep them pressed firmly to the floor, therefore the forefoot is held independently fixed and can follow neither a pronatory nor a supinatory twist occurring in the tarsus (Fig 121)

III. THE PATHOMECHANICS OF THE FOOT

A. THE PRONATED FOOT

1 **The joints**

In the *subastragaloid* joint the disalignment is caused by the inability of the ligaments and muscles to resist adequately the pronatory effect of gravity on the subastragalar articulation. The position of pronation which exists in this articulation forces the head of the astragalus downward forward and inward with the result that the arch of the foot is flattened and shows a convexity of the inner border The *tibio-astragalar* joint which moves only in the sagittal plane, in direction of flexion and extension, is not involved in the distortion In the *midtarsal* joint a rotation takes place in opposite direction to the pronation in the subastragalar joint a compensatory supination. The cause of it is the counter pressure of the floor upon the ball of the foot.

It is an error to assume that the pronated flatfoot is pronated throughout its entire length Due to the pressure of the floor the forefoot does not follow the pronation which the backfoot has assumed in cases of flatfoot (A Steindler[40]) There is a counter torsion between the backfoot and the forefoot in longitudinal

direction The forefoot, being fixed to the floor by the superincumbent weight, does not actually go into supination It is simply the back part of the foot which goes into pronation and, not being followed by the front part of the foot, the latter maintains a supinatory position relative to the backfoot We call this the *compensatory supination of the forefoot*"

Under the pressure of weight a break occurs in the midtarsal joint which does three things It produces the compensatory supination of the forefoot, described above Secondly, it permits the forefoot to be pressed up ward in dorsiflexion relative to the backfoot, which is then plantar flexed in respect to the forefoot Thirdly, it permits the forefoot to go in abduc tion relative to the back portion of the foot (Fig 122) In short we see that in the different joints the front foot is rotated against the back part in all three planes in which movement in midtarsal joint is possible The part of the foot distal to the joint is *supinated* against the pronated back part the front part is *dorsiflexed* against the plantar flexed back part and it is *abducted* against the ad ducted proximal portion of the tarsus



FIG. 122 Static flatfoot.

All the skeletal changes of the static flatfoot from the mildest to the severest degree are explained by these distortions

2. The weight distribution

The usual pattern of weight distribution is considerably altered in the case of the pronated foot Normally the line of gravity is behind the ankle joint during the restraining phase of the support period It then moves forward from the heel along the outer portion of the foot until it stands vertically over the ankle joint, when the propelling phase begins It then moves in a curved line medially to reach the first metatarsal head from which the take-off is enacted (D J Morton[11])

In the flatfoot this pattern is considerably changed The contracture of the tendo achillis forces the foot into valgus position and causes the line of gravity to run through the medial portion of the heel or even medially to it, behind the inner malleolus as the foot is set on the ground Because of the pronation, the progress of the line of gravity does not occur along the outer border but it travels forward to the big toe along the inner border of the foot. That deprives the foot of the important pronatory twist just before the take-off and greatly weakens the force of propulsion

B THE RELAXED TRANSVERSE ARCH

A relaxed and depressed transverse arch prevents the free flexor action of the toes and thereby also impedes the take-off and the propulsion The protrusion of the metatarsal head against the ball of the foot results in contraction of the extensors of the toes and in clawfoot deformity

IV THE CLINICAL PATHOLOGY AND DIAGNOSIS OF THE FLATFOOT

Three factors facilitate the early diagnosis of the static flatfoot.

1 The pain caused by the strain of the soft structures usually precedes the deformity

2 Because the soft structures under strain are highly endowed with sensory fibers, pain becomes strictly circumscribed and develops characteristic trigger points These points make it possible to identify accurately the structures under stress.

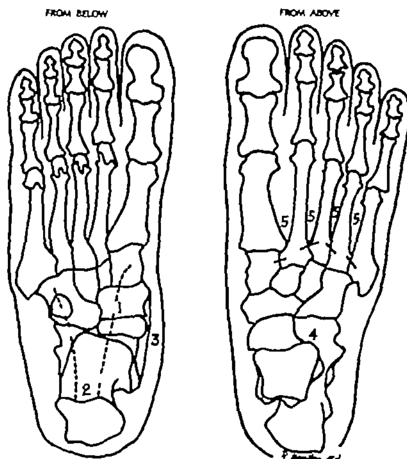


Fig 123 Trigger points of pain in the static flatfoot. 1) Trigger point for the medial border of the plantar fascia. 2) Trigger point for the insertion of the plantar fascia into the calcaneus. 3) Trigger point for the calcaneoscaphoid ligament. 4) Trigger point for the sinus tarsi 5) Trigger point for the intermetatarsal ligaments.

3 Disalignment is at first transitory, appearing only under weight bearing Only much later it becomes fixed and persists when the foot is at rest

A THE FUNCTIONAL DEFICIENCY PAIN

1 Spontaneous

Pain or ache in the foot is the most common complaint, it is accentuated by standing or exertion and is usually relieved by rest

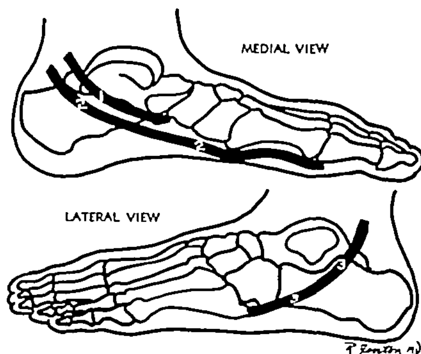


FIG. 124 Areas of tenderness in the static flatfoot. 1) Tenderness over the tibialis posticus. 2) Tenderness over the flexor hallucis longus. 3) Tenderness over the peronei tendons.

2. Pressure pain, ligamentous (Fig 123)

A pressure point at the inner border of the plantar fascia signifies strain of the plantar fascia.

A pressure point below the scaphoid, between it and the sustentaculum tali indicates strain of the calcaneoscaphoid ligament caused by the downward and forward push of the head of the astragalus

A pressure point at the anterior border of the lower surface of the heel suggests stress at the origin of the short flexors of the toes

A pressure point below the tip of the external malleolus denotes impingement of the tissues in the sinus tarsi as the foot goes into valgus position under weight bearing

3 Pressure pain, muscular (Fig 124)

The static muscle strain manifests itself by a diffused and poorly localized tenderness of the muscle, quite in contrast with fascial and ligamentous static strain which develops definite trigger points

Strain of the tibialis anticus produces a diffuse tenderness of this muscle at the anterior surface of the leg lateral to the tibia. Strain of the tibialis posticus elicits tenderness behind the posteromedial border of the tibia along the medial side of the leg Strain of the flexor of the big toe can be ascertained by following the course of the muscle under the big toe and the first metatarsal upward to the postero-internal surface of the leg Gastrocnemius strain causes tenderness along the gastrocnemius at the posterior aspect of the leg from the heel up to mid-calf (Fig 124)

B THE MORPHOLOGICAL CHANGES

The deformities are first transitory, that is, they appear only under weight bearing. Certain types of feet are particularly characterized by a natural laxity of the ligamentous and muscular apparatus and go promptly into a position of valgity when weight is borne. Usually the flatfoot deformity has existed since childhood but caused no incapacity in the earlier years Often one finds on examination that the heel cord is short and dorsiflexion in the ankle joint is restricted if the foot is placed in varus position Other components of the static flat foot are the pronation of the back part of the foot, the compensatory supination, the abduction and the dorsiflexion of the forefoot in the midtarsal joint. The entire foot appears strongly rotated externally in relation to the leg and the downward forward and inward displacement of the astragalus causes the bulge at the inner side of the feet with resulting convexity of the inner border The inner malleolus is prominent, and the lateral appears effaced there is considerable impingement of the sinus tarsi (Fig 125)



Fig. 125 Markedly pronated Static flatfeet.

Harris and Beath [1] found in x ray studies of this type of hypermobile nonfixed flatfoot that one of the essential features is the variation in the support of the body of the astragalus by the sustentaculum of the os calcis and the relation of the head to the anterior process of the calcaneum

If the head of the astragalus is not resting on the anterior process of the os calcis and is displaced medially, a large area of the bone remains un supported. In cases of hypermobile feet in which the anterior margin of the sustentaculum does not reach far enough forward to support the neck, these authors believe that this change in the relationship of the bones is even of greater importance in causing the deformity than is the deficiency of muscle power The fact is that in early stages of flatfoot before there are any fixed deformities the longitudinal arch flattens under the weight bearing and the astragalar head rests upon the relaxed and distended calcaneoscaphoid ligament, pushing itself forward downward and inward. This produces not only a flattened arch but also a convexity of the median border of the foot.

C THE PERMANENT OR FIXED DEFORMITY OF THE STATIC FLATFOOT

1 The spastic flatfoot

The foot becomes fixed by contracture of the soft tissues It no longer regains its normal shape when weight bearing is eliminated The fixed deformity is first due to the spasticity of the extensors and pronators of the foot, the reflex reaction to the painful ligaments is muscular spasm Gradually there is an adaptation of these contracted muscles and of the ligaments to the pathological position, so that the deformity sooner or later becomes fixed by actual muscle shortening and ligamentous contracture The contracted muscles are the peronei and the extensors of the toes

2. The rigid flatfoot

Skeletal deformities develop gradually by adaptation of bones to the articular disalignment. The product of this adaptation is the osseous flatfoot The plastic bones of the young patients are more susceptible to adaptive changes which are most conspicuous in the astragalus and the scaphoid The adult bone responds to stresses more with reactive osteoarthritic changes and marginal osteophytes These changes are most marked at the anterior edge of the ankle joint and the upper margin of the astragaloscaphoid articulation

V THE TREATMENT OF STATIC DEFORMITIES OF THE FOOT

A CONSERVATIVE

The objectives are *First,* to restore the foot to normal form at rest and to preserve this form on weight bearing by mechanical means *Second,* to restore the normal tone and tension of the soft tissues muscles and ligaments, which normally maintain the form of the foot when under gravital and other stresses

1 The relaxed flatfoot

The foot offers no resistance to correction It assumes deformation only under weight bearing The problem is therefore only one of maintaining cor rection This is done by mechanical devices placed in or on the shoe

a) The Shoe

The shoe must have a straight last, a rigid or semi rigid shank and a round toe. It has the proper relation between toe-ball and heel ball length, that is, the place for the big toe joint is not too far backward, as is often the case when the arch part of the shoe is too short and the toe part too long Individual feet vary greatly in this respect The shoe should fit snugly in the heel, and it should be wide in the toe so as to give ample room for the anterior arch and the free play of the toes The height of the heel is determined by the length of the heel cord.

For men 1 to 1¼ inches, and for women 1¾ to 2 inches is the usual height. The heel should be broad, as is the so-called Cuban heel Heels with tapered points are not to be recommended The firm sole protects the foot from the unevenness of the ground and provides a proper foundation for the arch support.

To strengthen the supportive function of the shank of the shoe a Jones' triangular heel can be applied It is an elongation of the medial side of the heel made by jutting it forward in triangular fashion to the level of the astragalo-scaphoid junction

b) The Support of the Longitudinal Arch

This is a mechanical device to fill the dead space between the sole of the shoe and the natural non weight bearing longitudinal arch. So-called inbuilt arches which are nothing more than a stiff inner counter are wholly inadequate for that purpose because they do not attach themselves closely to the sole of the foot and furnish no support for it.

In the construction of the insoles several points are to be observed The arch of the foot is not a part of a circle It rises gently from the ball of the big toe to a point just in front of the heel From here it slopes abruptly downward toward the heel The apex of the arch therefore is not at the middle of the foot but at the posterior half To fit an insole accurately it is best to use a plaster mold made with the patient sitting down

One can design the insole fairly accurately from an imprint on which the contours of the foot are outlined One then marks the ball of the great toe, the anterior contact point of the heel and the calcaneocuboid junction Into this area one then draws an outer line girding the ball of the foot and projecting tongue like toward the center of the anterior arch. It then circles along the lateral contour of the arch and ends medially at the anterior contact point of the heel According to the height of the arch two or three parallel inner lines follow the outer line This is done in such a way that, corresponding to the gentle anterior and the abrupt posterior slope of the arch, the distance between these lines is greater in front than in back similarly as various slopes are indicated in map drawing The contour lines then represent the successively smaller layers of insole which are stacked upon each other This gives the finished insole the gradual incline in front and a more abrupt incline in back. The basal layer as well as the smaller superimposed ones has a cut-out which allows the ball to rest firmly in the shoe with the forefoot in pronated position We use for these layers either cortex, a hard rubber substance, or solid leather

A very commonly used support is the metal footplate of Whitman it gives excellent support and prompt relief especially in acute arch strain

It remains now to correct the valgity in the astragalocalcaneal joint and the compensatory supination of the forefoot. For this purpose the heel of the shoe is supplied with a wedge of ⅛ to ¼ inch on the inner and the toe part with a ⅛ inch wedge on the outer side These cleats will reverse the length torsion of

the foot so that the back portion turns from the valgus into a varus, while the forepart is changed from a supinatory to a pronatory position That this counter twist restores the arch of the foot can easily be demonstrated on any pliable foot Placing the heel in pronation and the forefoot in supination flattens the longitudinal arch, conversely, placing the heel in supination and the fore foot in pronation markedly heightens the arch of the foot It occurs only in small children that the entire foot is in pronatory position from heel to toe Here the cleat is applied full length to the inner side of the shoe

c) Rehabilitation of Muscles and Ligaments

In the treatment of the relaxed flatfoot muscle re-education is instituted from the start A set of selected exercises designed to develop especially the tibiales muscles and the flexors of the toes is taught the patient to be carried out at home together with other activities which are capable of increasing the muscle tone of the legs An exception to this program are the acute cases of static flatfoot. The feet are swollen, tender and sometimes red, simulating an inflammatory condition They are unable to bear weight even with the help of insoles The most acute of these cases have to spend some time in recumbency until, under rest and treatment with heat and massage the swelling has disappeared Some may even require immobilization in plaster to obtain relief Pre-existing circu latory difficulties greatly retard the recovery, and it must be emphasized that every examination for flatfoot must include a thorough check on the circulatory condition The pulsation of the dorsalis pedis and posterior tibial arteries must be tested An oscillographic record should be taken on all cases in which a suspicion of vascular deficiency exists In cases of this type contrast bath and positional exercises should precede the ambulatory treatment.

A source of diagnostic error is the atrophic arthritis Swelling and pain of the joints of the foot and especially of the metatarsophalangeal articulations are not infrequently the presenting symptoms Other joints in particular the proximal interphalangeal joints of the fingers, should be carefully investigated

2. Treatment of the fixed deformity (rigid flatfoot)

If the condition is due to muscular spasm, it is useless to apply an insole or wedges on the shoe The form must be restored by manipulation, under anes thesia if necessary Then a plaster cast is applied for two to four weeks with the foot in complete correction and the arch well molded When the rigidity has disappeared and the foot has become pliable, insoles and cleats are applied and supination of the back foot is further secured by a T strap brace In some contractural flatfeet the rigidity is so great that it does not yield to manipulation under anesthesia It then becomes necessary to lengthen the contracted peroneal or even the extensor tendons before correction is obtained and a cast is applied A structurally shortened tendo achillis requires lengthening Re-education of the muscles begins as soon as the cast is removed

B OPERATIVE TREATMENT OF FLATFOOT

The conservative treatment of the static flatfoot is abandoned only under special conditions There are cases of chronically relaxed flatfeet in which all attempts to rehabilitate the musculature and to restore active balance fail and in which the deformity recurs as soon as the support is removed. Another type is a rigid flatfoot in which skeletal deformities make the restoration of form impossible A third type requiring operative procedure is the painful, rigid flatfoot in which weight bearing is not tolerated and which presents secondary arthritic changes The choice of operation depends upon the type of flatfoot.

The chronically relaxed pronated foot. Kidner's operation consists in the removal of the so-called prehallux or accessory scaphoid and the transposition of the posterior tibial tendon. R R. Fitch and B B King's[a] procedure is a transposition of the anterior and posterior tibialis muscles, and the method of Milch[a] is a fasciodesis between inner malleolus and the sustentaculum tali which places the foot in position of supination In general, operations on the soft parts alone do not quite safeguard the foot when weight is borne. Our procedure of choice is a combination tendon transposition with a cuneiform scaphoid fusion

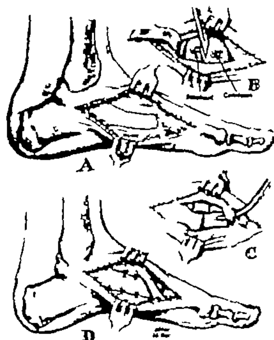


Fig. 126 Miller's operation for static flatfoot. (From P Lewin *The Foot and Ankle* Philadelphia, Lea and Febiger 1941 Fig. 93)

Miller's[a] operation consists in making a rectangular osteoperiosteal flap in the region of the first cuneiform toward the scaphoid. The flap has its base backward and is raised, then the articulation between scaphoid and cuneiform is resected The tibialis anticus tendon is sutured as far under the foot as possible thereby increasing the arch of the foot. The flap is then drawn strongly forward over the tibialis anticus tendon and sutured in place (Fig 126)

If the tendo achillis is short, it must be lengthened The foot is placed in plaster for six weeks A period of massage and exercise follows (Figs 127, 128)

In severer cases of relaxed flatfoot we found the arthrodesis of the Miller operation insufficient because it did not control the pronation It could be controlled only by a triple arthrodesis (Fig 129)

Most osteoplastic operations for flatfoot are based upon the reconstructive







FIG. 127 FIG. 128

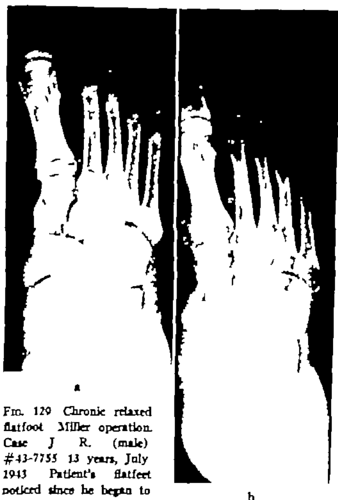
FIG. 127 (*upper left*) Chronic relaxed flatfoot Miller operation. Case E. N (male) #45-6644 12 years, *July 1945 Patient always had flatfeet and a peculiar gait. Three months before admission he* complained of aching feet which were relieved by rest could not walk far without pain. Examination showed marked valgus deformity of both heels no pressure points Miller operation performed on both sides with lengthening of the tendo achillis July 23 1945 When seen again, March, 1946 he had no complaint and was able to walk without trouble feet well corrected.

FIG. 128 (*upper right*) Chronic relaxed flatfoot Miller operation Case H H (male) #43-8536 14 years, August 1943 Patient always had flatfeet Symptoms began 13 months before admission after a fall 14 feet complained of pain in both feet. Examination showed marked valgus, abduction of the forefeet and marked depression of the longitudinal arches spasm of the peronei left and tenderness over the sinus tarsi and spring ligaments. Miller operation on left side performed *June 22 1944 following operation patient entirely relieved. When seen last, in May 1946 almost* two years later patient was free of symptoms.

work of Hoke,[17] who introduced the principle of arthrodesing the scaphocuneiform rather than *the astragaloscaphoid, thereby preserving the function of the* midtarsal joint

There remains the inveterate and severely deformed flatfoot in which tendon transference and limited arthrodesis are inadequate These cases require triple arthrodesis and wedge resection to correct the deformity A subastragalar fusion through a posterior approach by insertion of a bone graft into a channel made between the adjacent surfaces of the os calcis and the astragalus is described by Gallie.[8] Harris and Beath[11] approach the astragaloscaphoid joint as well as the joint between the sustentaculum and the neck of the astragalus from the medial side, fusing the joint by removing the articular cartilage and implanting cancellous bone grafts In all cases of short tendo achillis lengthening of the cord is performed as part of the operative procedure

Statistics on the operative treatment of static flatfoot (Greteman[*]) The end result in 17 cases of cuneiform scaphoid fusion with transplantation of the tibialis anticus into the scaphoid was excellent in 11 and good in four, totalling



a b

FIG. 129 Chronic relaxed flatfoot Miller operation. Case J R. (male) #43-7755 13 years, July 1943 Patient's flatfeet noticed since he began to walk complains of pain along longitudinal arches and in calf Examination showed marked valgus deformity flattening of the longitudinal arches and prominence of the tuberosity of the scaphoid tenderness on pressure over the longitudinal arch and right tendo achillis. Miller operation performed on right foot August, 1943 same operation on left foot November 1943 Following surgery patient given solid leather insoles when seen one year later was walking with such a valgus deformity that a triple arthrodesis was indicated, but there was no pain. a) Before. b) After Miller operation.

88.2 per cent satisfactory results Of 15 cases of cuneiform scaphoid fusion with transplantation of the tibialis anticus and lengthening of the tendo achillis good and excellent results were obtained in 13 cases, or 86 6 per cent. On the other hand tibialis anticus transplantation with or without tendo achillis lengthening and without fusion gave poor results in all 6 cases.

VI DISABILITIES OF THE TRANSVERSE OR METATARSAL ARCH OF THE FOOT

A ANATOMY

The transverse arch is formed by the heads of the five metatarsals united by the dorsal and plantar interosseous ligaments The term anterior metatarsal arch is not quite correct for the reason that when weight is borne, the curve of the arch disappears and the arch lies flat on the ground The transverse arch is supported not only by the ligaments mentioned above but also by the flexor muscles of the toes and the intrinsic muscles of the foot When the muscles relax from over use or disease, it falls upon the ligamentous structures to maintain the arch There are some anatomical reasons why this relaxation of the arch occurs easier in some persons than in others (Fig 130)

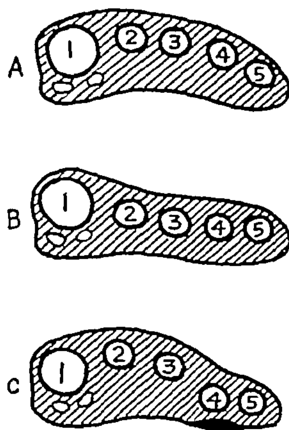


Fig. 130 Schematic representation of metatarsal arch. A) Normal position of metatarsal heads and sesamoid bones. B) Depressed metatarsal arch. C) Depression of fourth metatarsal head with production of callosity (From P Lewin *The Foot and Ankle* Philadelphia, Lea and Febiger 1947 Fig 116)

A congenitally high longitudinal arch is predisposed to metatarsal arch strain Another predisposing factor is a congenital variation called Morton s syndrome [33] It consists of a short first metatarsal, a thickened shaft of the second metatarsal, a hypermobile first metatarsal and a proximal displacement of the sesamoid of the first metatarsal The principal mechanical implication of this syndrome is that the short first metatarsal causes a concentration of stress on the second metatarsal so that a heavier load falls upon the articulation of this metatarsal The shaft of the latter becomes thickened under the increased stress

The hypermobility of the first metatarsal is of mechanical importance because it prevents the first metatarsal from engaging the ground firmly, and the brunt of the weight remains on the second metatarsal The proximal shift of the sesamoid actually shortens the first metatarsal because the sesamoids are the point of contact with the ground

B THE CLINICAL DIAGNOSIS

1 Pain

On exertion. Non neuralgic. The stress on the transverse arch produces

pain over the volar aspect of this arch, behind the metatarsal heads The pain disappears on rest. It is relieved by pressure above the metatarsal heads, which restores the arch, or by lateral compression of the metatarsal shafts which has the same effect.

On exertion. Neuralgic. The *relaxation of the intermetatarsal junction* causes compression of the common digital nerve between the third and fourth metatarsals This referred pain appears on pressure at the metatarsophalangeal articulation The pain characteristically radiates to the tip of the toe It is usually the third and fourth toes which show this symptom, known as Morton's toes first described by T G Morton[11] (1875) It usually yields to mechanical support which relieves the pressure from the head of the fourth metatarsal. Morton himself treated resistant cases by excision of the metatarsophalangeal joint and the soft tissue including the nerves around the joint.

Spontaneous. Neuroma. The metatarsalgia is often due to the formation of a neurofibroma or angiofibroma in the course of the digital nerve (Mc Elvenny[10]) It has a rather constant location between the third and fourth toes, a little proximal to the metatarsal head Hoadley (1893), reporting on six cases of metatarsalgia which failed to respond to conservative treatment, exposed the digital branches of the lateral plantar nerve through the fourth toe and found a *small neuroma The removal of this neuroma produced a prompt* and perfect cure In 1940 the exact pathological basis and the rational surgical treat ment were established by Betts,[2] followed in this country by McElvenny[10] They found a tumor situated on the lateral branch of the sensory nerve which *supplies the third and fourth digits. The condition is characterized by severe* paroxysmal pain usually arising beneath the heads of the third and fourth metatarsals and extending to the opposing sides of the third and fourth digits Sometimes the pain radiates up to the posterior leg as far as the hip Firm pressure over the site of the neuroma causes radiating pain as well as paresthesia

Grossly the neuroma represents itself either as a pancake-like thickening or a fusiform swelling of the nerve trunk. The early nerve changes consist in interstitial edema spotty in character associated with irregular demyelinization and swelling of the entire nerve trunk. This is accompanied or shortly fol lowed by rather marked proliferation of the neurolemma nuclei which, like the edema is also spotty or focal in distribution The question arises, why the *fourth digital nerve should be the one selected for development* of this neuroma. This nerve is unlike its neighbors, of double derivation, being formed by anastomotic branches from both medial and lateral plantar nerves The arch which is described by the two constituent branches forms a transverse link across the outer or superficial aspect of the flexor brevis digitorum muscle The fourth digital nerve is more or less anchored posteriorly and lacks the mobility of the other digital nerves Stretching of this nerve occurs in dorsi *flexion of the foot and toes and is intensified in walking*

2. Contractures

As a result of the depression of the anterior arch, the tension in the long and short extensors of the toes is increased Active and passive volar flexion is impeded A clawfoot deformity develops in severer cases with secondary hammer toe deformity in the proximal interphalangeal joints

Purely static relaxations of the metatarsal arch must be distinguished from several other conditions with a similar clinical picture *Arthritis* of the metatarsophalangeal joint produces metatarsalgia often as the presenting symptom. Depression of the transverse arch is a symptom of circulatory lesions such as endarteritis or thromboangiitis obliterans Sometimes an inflammation of a metatarsal bursa produces swelling and soreness at the ball of the foot which might be mistaken for a purely static disorder There are several bursae in the metatarsophalangeal articulation (Hertzler[11]) The most common is at the metatarsal heads and around the flexor tendons in the metatarsophalangeal region

C THE TREATMENT OF ANTERIOR METATARSALGIA

1 Conservative

In acute cases the treatment is rest, heat and massage or, for immediate relief, temporary strapping of the anterior arch. When the acute symptoms have subsided the essential point is the support of the anterior arch during weight bearing. The point of support is behind the heads of the metatarsals, but it is more effective to construct the insole so that it supports the longitudinal arch also and thereby relieves the metatarsal arch entirely from body weight The weight is then concentrated over the instep, and the ball of the foot is completely relieved of pressure In addition an anterior crossbar or anterior heel placed across the front part of the foot just behind the metatarsal arch eliminates pressure on the ball of the foot Hauser's[12] so-called comma bar and Lewin's[13] so-called metatarsal crescent serve this purpose

2. Operative

a) The Relaxed Anterior Arch

The occasional intractible case of anterior arch relaxation requires operative interference Krida[11] performs an osteotomy of the first and fifth metatarsal bones so as to mobilize the metatarsal shaft and then places a long piece of fascia around the metatarsal necks, under the extensor tendons on the dorsal surface, under the flexor of the big toe, over the common flexor of the toes and again under the flexor of the little toe The two ends of the strip are then drawn tightly together to secure the arch

4) The Plantar Neuroma

Conservative treatment should always be given a preliminary trial The irritation of the nerve may be stopped by shoes of sufficient length and width.

External transverse bars or suitable padding within the shoe may relieve the symptoms If the symptoms persist, however, they are best treated surgically A plantar incision between the heads of the metatarsals will expose the nerve which can be felt to lie just beneath the plantar fascia and below the deep transverse metatarsal ligament. This can be exposed easily, and a wide resection can be carried out through this incision. The proximal end of the nerve can be severed well back into the short muscle of the foot, thereby avoiding future irritation of a section neuroma

c) THE FIXED DEFORMITIES, CAVUS AND CLAWFOOT

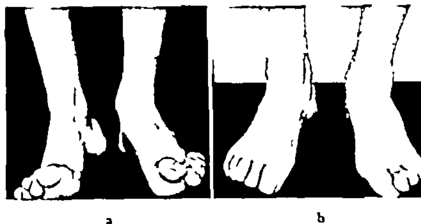
The depression of the plantar arch is often associated with fixed retraction of the toes and cavus deformity with contracture of the plantar fascia. Moderate cases which are relieved by conservative measures of their subjective symptoms require no further procedures The problem is the advanced case of hammer toes or cavus deformity A hammer toe may be due to heredity, but more often it is the result of mechanical stress such as occurs in a depressed anterior arch The essential feature is that the contracture of the flexor tendons produces a fixed deformity at the proximal interphalangeal joint. A knuckle develops on top of the joint and is usually occupied by a rather painful corn or callus. The volar portion of the capsule of the joint is contracted. The toes most often involved are the second and fifth toes The second hammer toe is particularly important, because it is often combined with a hallux valgus

We believe it is a mistake to amputate hammer toes, except the fifth toe The operation of choice should be resection of the midphalangeal joint of the toe combined with tenotomy of the flexors Tenotomy of the tendon alone, even with operation on the capsule, is seldom sufficient. It is better to resect the midphalangeal articulation and straighten the toe, holding it in this position sufficiently long to secure good fusion.

Claw toes In extreme cases the retracted toe actually rides on the neck of the metatarsal A satisfactory procedure for this situation is that of Hoffman.[18] It consists in resection of the metatarsal head from a volar transverse incision (Fig 131)

Needless to say, following such a radical operation the patient must wear a permanent support. He is not allowed to bear weight for at least four to six weeks after the operation and then only in a shoe properly provided with an insole and an anterior bar to relieve pressure from the ball of the foot. Our statistics on this operation (A W Ciani, 1937) show that in 15 cases the end result was good in 64 per cent and fair in 16 per cent.

The *cavus* deformity offers greater difficulties to correction If it is of skeletal nature, simple wrenching combined with operation on the soft structures hardly suffices *Skeletal cavus deformities* require osteotomy and wedge resection in the region of the midtarsal joint. These are formidable operations which should be undertaken only in the face of marked and persistent complaints and after conservative trials to relieve the highly sensitive anterior arch



a b

Fig. 131 Hoffman Operation for Cavus and Clawtoes. Case O H #38-15370, 39 years, March 1938. Patient complained of pain on ball of foot for over 20 years, aggravated by standing and walking. Examination showed marked claw deformation of the toes and bilateral hammertoe with metatarsal heads protruding sharply at the plantar surface. Hoffman operation performed on left foot March, 1938, and on right foot in October 1938 together with excision of inter phalangeal joint of second toe on both sides. The third and fourth toes were fused in the same manner November 1938, in order to correct hammertoes followed later by resection of first and second metatarsal heads of right foot. During observation of one year patient had good correction and was without pain. a) Before. b) After operation.

have failed. In most cases of cavus deformity one will find the plantar fascia strongly contracted. As a preliminary step to the correction, the fascia is stripped from the os calcis (A. Steindler[11]) (Fig 132) The plantar fascia can easily be reached from a small incision over the inner aspect of the os calcis It should be incised where it blends with the lower surface of the os calsis and should be stripped off bluntly so as to leave the periosteum on the os calcis and prevent reformation of periosteal bone

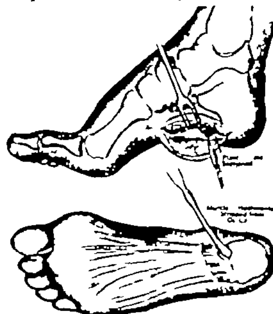


Fig. 132 Steindler stripping operation of plantar fascia. (From A. Steindler Orthopedic Operations, Springfield Charles C Thomas, Publisher 1940 Fig. 34)

After the fascia is stripped and the foot is wrenched in cases showing no fixed skeletal deformity the anterior arch can be raised by tendon transposition The procedure of R Jones and Sherman consists in the section of the long extensor tendons of the toes at the metatarsophalangeal joints and their insertion into the necks of the metatarsals Hibbs operation inserts the extensor tendons into the lateral cuneiform

VII. THE HALLUX VALGUS

A THE PATHOLOGY

Except for a certain congenital deviation of the big toe, the usual type of hallux valgus is of static nature and is accompanied by depression of the anterior plantar arch The deformity consists in an outward deviation of the big toe, prominence of the big toe joint, and an exostosis at the medial surface of the metatarsal head A bursitis develops over the big toe joint and, in addition, the sesamoid bones are displaced so that the lateral one comes to lie in the inter metatarsal space.

There are three stages of development (Verbrugge[13]) In the first stage the hallux valgus manifests itself by the change in the relationship of muscles and bones only The first cuneiform is slightly displaced inward and forms a small but visible angle with the second cuneiform. The first metatarsal is in abduction relative to the axis of the foot (metatarsus primus varus), and the head of the metatarsal is subluxated inward and forms a protuberance under the skin. The long extensor slides off the first metatarsal and forms the base of a triangle, the apex of which is the articulation, and the sides, the first metatarsal and the big toe. The toe itself is rotated in pronation The long extensor is tight. The sesamoids are displaced laterally together with the muscles inserted into them—the abductor and the short flexor inserted into the medial, the adductor inserted into the lateral sesamoid Khoury[19] found this displacement in 143 of his 232 cases, or 61 6 per cent.



FIG. 133 Hallux valgus.

In the second stage the skin over the head of the first metatarsal becomes sclerotic and thickened and is covered with callus The bone reacts to pressure with formation of an exostosis Occasionally a serous bursa of different size develops The osseous prominence together with the serous bursa and the callus constitute the characteristic bunion The joint capsule shrinks in its lateral portion causing the hallux valgus deformity to increase to as much as 90 degrees. The big toe still can be straightened manually, but if released it returns to its position of deformity

The third is the stage of the secondary lesions Arthritic changes appear in the metatarsophalangeal joint. The articular cartilage of the head of the metatarsal is worn off and osteophytes develop (Fig 133)

B THE CLINICAL PATHOLOGY

While the truly congenital case is rare (only 0 5 per cent, Khoury), the hereditary tendency to this deformity has been estimated as high as 50 per cent (Sandelin[35])

The subjective symptoms begin rather insidiously The patient complains of pain at the inner border of the articulation of the big toe, sometimes aggravated by weather changes As the hallux valgus becomes more marked, the pain becomes more violent It follows the nerve tracts of the internal sensory branch which penetrates the bursa and traverses along the inner border of the foot Compression of the shoe and its effect on the serous bursa, particularly when irritated by secondary inflammation, and the arthritis established in the metatarsophalangeal joint explains the pain

The adduction of the first metatarsal favors the flattening of the transverse arch, so that the forefoot appears considerably broader (splayfoot) One observes this broadening in persons with ligamentous weakness, before it comes to an actual deviation of the big toe A similar deformity of the little toe with prominence of the metatarsal head and exostosis is known as tailor's bunion

The x ray signs. The x ray shows peculiar changes of the first metatarsal Its shortness is one of the features of Morton's syndrome [32] The second metatarsal is longer and becomes the principal point of support of the foot This fact has a great deal to do with the flattening of the anterior arch which accompanies the hallux valgus The pronation of the first metatarsal causes the sesamoids to be displaced outward and laterally, so that the internal sesamoid is entirely covered by the head of the metatarsal, while the external appears in the first interosseous space The latter is often bipartite One also recognizes the obliquity of the articular line between the first cuneiform and the first metatarsal (Berntsen[1]) Khoury[19] observed it in 40.5 per cent of hallux valgus cases Accompanying valgus deformity of the fifth metatarsal was found in 5.2 per cent (Khoury[19]) Arthritic changes at the heads of the metatarsals are noted in farther advanced cases

C THE TREATMENT OF HALLUX VALGUS

1 Conservative methods

These are designed to give relief and comfort but they cannot claim any effect on correction of the deformity, which is an operative problem Still conservative methods have a definite place Certain conditions make operative interference inadvisable, for instance a poor general systemic condition or circulatory disturbance such as thromboanglitis obliterans In milder cases results can be obtained by correction of the pronated foot and by restoration of the anterior arch This is accomplished by arch supports and massage and exercise An elastic bandage 5 cm wide or a leather cuff over the shafts of the metatarsals (Hohmann [8]) relieves pain caused by anterior arch depression So far as the hallux valgus is concerned corrective devices worn during the night

such as wedges of rubber or felt, gauze or cotton between the first and second toes are useless except in very young children

2. Operative treatment of hallux valgus

In his treatise on hallux valgus Khoury[19] enumerates not less than 60 operations devised for this deformity One of the oldest goes back to Hueter (1877), and in modified form it is still used today Of all these operations we select a few now generally accepted methods and shall formulate the specific indication for each procedure The operations may be divided into those of the soft tissues such as ligaments and bursae, operations on bone such as osteotomy of the phalanges, and finally mixed operations which are carried out both on soft parts and bone

Simple extirpation of the exostosis was first recommended by Schede (1904), with presumably good results, although the procedure has no effect whatsoever on the deformity Nevertheless, as late as 1940, McElvenny[29] presented statistics of 100 patients treated by simple removal of the exostosis without correction of the hallux valgus There are cases in which such a simple operation may well be applied if the deformity is not too great and if most of the complaints are centered about the exostosis

Later Schede attempted a straightening out of the toe by tenotomizing the capsule at the lateral aspect as well as the adductor tendon. One of the more useful simple methods is that advocated by David Silver[34] (1923) He uses a curved incision around the metatarsophalangeal articulation, removes the bursa and then incises the capsule in a "Y" in order to form three flaps, one dorsal on plantar and one distal, the latter with its base at the proximal phalanx. With the toe in extreme abduction the lateral portion of the capsule is severed. The distal flap is then dissected, and the correction of the deformity is accomplished by pulling the distal flap strongly upward and fastening it on the inner side of the articulation This method is still widely used and is very practical for lesser degrees of hallux valgus

Payr (1925) added to the excision of the exostosis the dissection of the adductor the opening of the capsule and ligaments on the outer side and finally the removing of the cartilaginous exostosis a method which is very similar to that later devised by McBride.[27]

Finally as already mentioned, Hueter (1877) first practiced resection of the head of the first metatarsal Thirty years later (1908) Mayo[28] revived this method resecting likewise the head of the first metatarsal but adding to it an arthroplasty by means of a pedunculated flap

To correct the metatarsus primus varus osteotomies were introduced. Ludloff's method is an oblique osteotomy in dorsoplantar direction The operation of Hohmann (1921) consists in an osteotomy directly behind the metatarsal head The transverse diaphyseal osteotomy of the first metatarsal in cases of metatarsus varus primus was again advocated by Hawkins, Mitchell and Hedrick (1945)

The principle of resection of the proximal phalanx of the big toe was first introduced by Keller (1904), who published three cases all having satisfactory results Later this was taken up by Brandes,[8] who resected the proximal two thirds of the basal phalanx Girdlestone's[7] method is simply a combination of

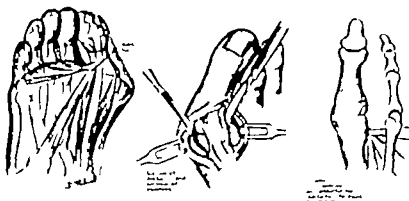


FIG. 134 McBride operation.

resection of the basal phalanx, already practiced by Keller (1905), with the oblique osteotomy of Ludloff and Mau

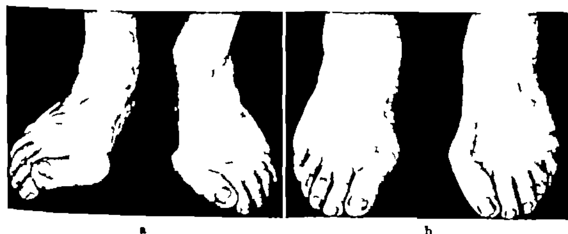
The principle of arthrodesing the metatarsal base with the cuneiform as well as removal of the exostosis, is incorporated in the method of Lapidus[32] Finally, Stein[41] sections the contracted soft parts, especially the adductor, removes the exostosis and performs a capsular plasty

Of all these operations we select three which we apply according to the merits of the case

1 In *milder cases* we use the operation of *Silver*

2 In *more extensive cases*, where there is a definite hallux valgus deformity but no arthritic changes we use the operation of *McBride* In cases in which this parallelism of the metatarsal cannot be obtained an *osteotomy* is added to the operation The osteotomy corrects the deviation of the first metatarsal (Figs 134 135 136)

3 In cases which show *arthritic changes* in the joint the choice is between



a b

FIG. 135 McBride operation. Case C. M. a) Before. b) After



FIG. 136. McBride operation. a) Before. b) After

two operations, the Mayo-Hueter bunion operation and the operation of Keller Brandes For this type of case the operation of Hueter Mayo is probably the most widely used. It is simple and effective and gives good results in cases of badly deformed and painful hallux valgus and in adult cases with arthritic lesions The disadvantage of the Hueter Mayo operation is that the principal point of support of the foot is removed from the longitudinal and



FIG 137 Mayo operation. Case I. H. #40-10483 61 years, August, 1940. Patient's feet began being painful four years ago condition became gradually worse. Right foot has a marked exostosis and hallux valgus, a hammer toe of the second, subluxation at the metatarsophalangeal joint and marked tenderness. *Left foot* has a hallux valgus with exotosis. X-ray showed arthritic changes at metatarsophalangeal joint. Bilateral Mayo operation performed August, 1940, and a Hoffman resection of the third and fourth metatarsal heads on the right in November 1940 End result four years later very satisfactory no complaint. a) Before. b) After

transverse arches, which becomes particularly noticeable in the gait (Fig 137) For this type of case we prefer the Keller Brandes operation which consists in resection of one fourth or one half the basal phalanx of the big toe If one-half the basal phalanx is removed, the risk of a postoperative rigidity of the joint is avoided (Fig 138)



a b

FIG. 138. Keller Brandes operation. Case H. J., #42 700, 57 years, January 1942 Patient complained of pain at big toe joint for several years. Examination showed bilateral hallux valgus with hammer toes on both feet and calluses over heads of metatarsals X ray showed arthritic changes. Keller Brandes operation performed on right foot, together with resection of second, third, fourth and fifth metatarsal heads and the interphalangeal joints of these toes. End result ten months later was good. a) Before. b) After

STATISTICS ON HALLUX VALGUS OPERATIONS (W R Hamsa[10])

Up to 1937 there were 339 cases of bunion operation available for study, with a minimum of two months and a maximum of 22 years' observation The results were as follows

Silver Operation 178 bunions
Good 57 per cent
Fair 22 per cent
Poor 21 per cent

Mayo Resection of the Metatarsal Head 126 bunions
Good 61 per cent
Fair 30 per cent
Poor 9 per cent

McBride Plasties 14 bunions
Good 36 per cent
Fair 50 per cent
Poor 14 per cent

Brandes Operation 21 bunions
Good 19 per cent
Fair 48 per cent
Poor 33 per cent

From 1937 and 1938 an additional 26 cases with 43 bunions were reviev (F E Thornton[43]) Two bunion operations by the Silver method were failu Of the cases with McBride operations 7 were fair and 21 good. All 12 ca operated by the Mayo procedure gave good results

VIII. THE OVERLAPPING FIFTH TOE AND TAILOR'S BUNION

The overlapping fifth toe is the counter part of hallux valgus on the late side of the foot. The toe is adducted toward the midline, is dorsiflexed in metatarsophalangeal joint and outward rotated so that the nail faces latera

The Lapidus[42] procedure is a transposition of the severed extensor tendon the little toe, in which the distal stump of the tendon is transplanted media and plantarly under the toe, being sutured into a slot made in the abduc of the fifth toe Others[8] simply expose the extensor tendon and lengthen adding a capsulotomy in order to correct the position of the toe We find tl in most cases of badly overriding fifth toe, it is just as easy and more effici to remove the toe entirely We have never seen any untoward effect from su a removal

IX. THE HALLUX RIGIDUS

The extension of the big toe is limited If weight is thrown forward onto t big toe joint, and especially at the take-off, a painful strain is produced. Ve often there is hypertrophy of the bone at the dorsal surface of the metatar head, the base of the distal phalanx may show similar ridges Conservative tre ment may try to relieve the strain from the big toe joint by a supporting ins and a metatarsal bar If this is not successful, an operative procedure is nec sary Simple excision of the osteoarthritic ridge at the head of the metatarsal the base of the basal phalanx is entirely insufficient. It is necessary to not on trim the portion of the head of its marginal exostosis but also to remove pa of the shaft of the basal phalanx so as to give free range to the extension the big toe For this purpose we prefer the Keller Brandes operation (Fig. 139

X. THE MARCH FRACTURE

The so-called march fracture is a condition in which a hairline fracture occu in one of the metatarsals following strain and stress "Fatigue fractures" ha been observed in other bones, particularly in the fibula, the radius and even tl neck of the femur

Full information on the history of this disability is contained in a compr hensive article by Leveton[3] It seems that a German military surgeon, Brei haupt, was the first to associate this injury of the foot with the trauma marching Since then numerous other observers have described similar cond

tions, but it was not until 1897 that Stechow demonstrated that this foot complaint was due to fracture of the metatarsal bone The condition is known also as Deutschlander disease (from a description in which this author ascribed the condition to a hematogenous bacterial periostitis) There is a general belief that this condition is due to the summation of protracted foot strain, but there are anatomical conditions which favor the development of this fracture Sirbu and Palmer[77] maintain that the march foot occurs in young soldiers unaccustomed to the rigors of long marches, but only in those whose feet are inherently weak or flat Of 259 cases of metatarsal march fracture Leveton[78] found that 105 had weak or flat feet, but the feet of 59 patients were perfectly normal, and



FIG. 139 Hallux rigidus resection basal phalanx. Case C McC a) Before b) After

17 had even a pes cavus In 76 the first metatarsal was shorter than normal, in 114 it was of normal length in 69 it was longer than normal

Symptoms. The condition develops insidiously with slowly increasing pain which first arises after prolonged, excessive effort and later after ordinary exercises The gradual relaxation of the intermetatarsal ligaments leaves the metatarsals unprotected. The strain affects principally the second and next the third metatarsal.

The x ray shows a hairline fracture through the cortex of the metatarsals and later a beginning callus formation Still later a spindle-shaped mass of bone is formed surrounding the lines of fracture The fracture is complete in the majority of cases when they come under observation, but there is no displacement of the fragment. According to Krause,[80] there are no x ray signs until seven to 10 days after the onset of symptoms Only swelling of the soft tissues is noticed From 10 to 17 days after injury there is a little periosteal fuzziness at the site of the beginning fracture, and on close observation a hairline fracture can be seen Later the periosteal shadow appears, and a more

circumscribed callus is visible Finally, the case ends with slight thickening of the cortex and solid union

Treatment is immobilization in plaster for four to eight weeks, or at least rest, hot application and relief from weight bearing Others recommend bed rest until pain and edema have subsided, and then complete immobilization by plaster applied with the foot in dorsiflexion and inversion

XI THE PAINFUL HEEL. THE HEEL SPUR

While various factors may produce heel spurs, such as infectious periostitis or metabolic disturbances, the painful heel is most often of purely static nature It is caused by excessive pressure upon the heel and is facilitated by improper shoes, heavy weight or static deformities of the foot, such as flat foot.

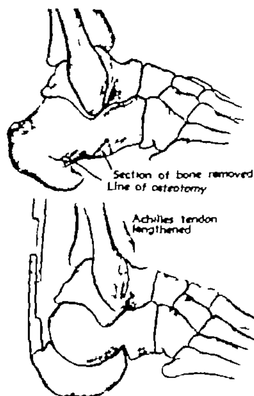


FIG 140. Rotation osteotomy of os calcis for resistant heel pain. (From A. Steindler Orthopedic Operation, Charles C Thomas, 1940, Fig. 89)

The site of the calcaneal spur is the inferior and medial aspect of the posterior process It is here where the plantar fascia takes its origin, and pressure applied to this area or the pull of the plantar fascia results in a slight separation of the periosteum In contrast to the inflammatory spurs, the static spur is not an apposition but part of the bone itself The stress lines of the spur are continuous with the trajectories of the body of the os calcis Inflammatory spurs show no such internal architecture

The symptoms of calcaneus spurs are pain, tenderness, swelling and limp The onset is gradual, and pain and tenderness are felt along the median border of the os calcis or at the attachment of the plantar fascia The x ray evidence is not always commensurate with the clinical symptoms Whether pressure symptoms develop depends much more upon the shape of the spur its sharpness and its downward direction

Conservative treatment should always be tried It consists in application of a very rigid support which grips the arch of the foot firmly and has a cupped out heel so that most of the pressure on the spur is eliminated If conservative measures fail operation becomes necessary The usual operative procedure is the removal of the spur from a medial incision alongside the os calcis All of the spur must be removed across the entire width of the os calcis Of a total of 71 con

servatively treated heels, we found 46 4 per cent had complete and permanent relief Only slight or no relief was found in 53 5 per cent The statistics[11] on operative removal show complete relief of symptoms after operation in 51 7 per cent, 41.3 had some postoperative recurrence of symptoms varying from mild to severe pain In extreme cases when removal of the spur has resulted in recurrences, we have applied the rotation osteotomy (Fig 140) This operation changes the point of contact for the os calcis by rotating the posterior process of the os calcis so that its lowermost point is deflected upward and forward, and the weight pressure is placed on the posterior instead of the inferior surface of the heel Of 8 cases so operated complete and permanent relief was obtained in 75 per cent

XII THE CALCANEAL BURSITIS

In 1928 Haglund described an anatomic variation in which the upper border of the os calcis is drawn out higher than normal and causes the shoe to press against it. The result is a hypertrophy of the skin and inflammation of the supracalcaneal bursa with an ossifying periostitis In order to relieve the condition a resection of the upper end of the calcaneus is performed

XIII THE INGROWN TOENAIL

Ingrown toenail is the name for a disturbance in which one or both of the lateral margins of the nail are turned down and imbedded into the soft tissue around the nail It produces an inflammation at the edges as the nail digs into the soft tissue and often causes fissures and warty growth in the inflamed skin The chief causes for this disability are short and pointed shoes or high heels, short and tight stockings or improper trimming of the toenails

Early cases may be treated conservatively by pushing the overhanging edges of the skin back and freeing the edges of the nail. In more advanced cases and especially if infection has set in, it is the practice to remove the lateral portions of the nail without destroying the matrix A very simple and efficient procedure is that of Newman which consists in inserting a very thin metal foil over the nail. The edges of the foil are curled under so that it can be slipped under the nail edge separating it entirely from the underlying bed

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